THE MEDICAL JOURNAL OF THE SOUTH-WEST

Incorporating
The Bristol Medico-Chirurgical Journal

PUBLISHED UNDER THE AUSPICES OF THE
BRISTOL MEDICO-CHIRURGICAL SOCIETY

Editor:
J. E. CATES
M.D., M.R.C.P.

with whom are associated
A. H. GALE, M.D., D.P.H.: *Assistant Editor*
J. MACRAE, M.D., D.P.H., F.R.E.P.S.
N. J. BROWN, M.R.C.P.
J. P. PARTRIDGE, F.R.C.S.

1953 - 1955 Vol. 70

J. W. ARROWSMITH LTD., WINTERSTOKE ROAD, BRISTOL

For
"M
Und
Me
The
Obs
Dif
Pha
Her
Her
Col
Dar
Pra
The
Col
A F
Mo
Boo
Dar
Cat
Son
"G
Col
Cou
Me

U of M Bindery
AUG 20 57

B610.5
B756

1955 Contents of Vol. 70

	page
Foreword. By Sir Philip Morris, C.B.E., M.A., LL.D.	1
"Marasmus". By A. V. Neale, M.D., F.R.C.P., D.P.H.	3
Underwater Breathing. By Ronald Woolmer, F.F.A.R.C.S.	9
Medicine at Home and Abroad. By John Apley, M.D., M.R.C.P.	16
The Elderly Chronic Sick	20
Observations on Blindness. By R. Ramsay Garden, M.A., M.B., Ch.B., D.O.M.S.	33
Diffuse Angiomatosis of the Small Intestine Causing Melaena. By John A. Vere Nicoll, F.R.C.S., D.A.	47
Phaeochromocytoma of Adrenal Gland with Paroxysmal Hypertension. By G. M. Colson, B.M., M.R.C.P	50
Herpes Zoster Encephalitis. By R. D. G. Creery, M.D., M.R.C.P., D.C.H.	54
Herpes Zoster Generalisatus. By Mark Hewitt, M.B., M.R.C.P.	57
College of General Practitioners, South Western Regional Faculty	61
Damned Drugs (Part I). By G. E. F. Sutton, *M.C.*, M.D., F.R.C.P.	71
Practical Points in the Home Treatment of Tuberculosis. By C. de W. Kitcat, M.R.C.S., L.R.C.P.	79
The Tropics on our Doorstep. By Sir George R. McRobert, *C.I.E.*, M.D., F.R.C.P.	83
Colonial Medical Research. By H. S. L. Heller, M.D., Ph.D., M.R.C.P.	91
A Radiologist Visits Africa. By J. H. Middlemiss, M.D., F.F.R., D.M.R.D.	95
Mongolism, Hypertrophic Pyloric Stenosis Treated with "Skopyl", and Death from Miliary Tuberculosis and Meningitis. (A Clinico-Pathological Report)	97
Book Review	102
Damned Drugs (Part II). By G. E. F. Sutton, *M.C.*, M.D., F.R.C.P.	103
Cat - Scratch Disease. By D. Bryant, M.B., B.Chir., and R. P. Warin, M.D., M.R.C.P.	113
Some New Methods of Radiotherapy. By R. C. Tudway, M.B., B.S., B.Sc., F.F.R.	116
"General Practice". By G. F. Abercrombie, V.R.D., M.D.	120
Colloid Cyst of Third Ventricle (A Clinico-Pathological Report)	127
Cousin Marriage. By J. A. Fraser Roberts, M.A., M.D., D.Sc., F.R.C.P.	142
Meningitis in Childhood. By Beryl D. Corner, M.D., F.R.C.P.	150

U of M Bindery
AUG 20 57
1601787

CONTENTS

	page
Meningitis. By A. M. G. Campbell, M.D., F.R.C.P.	162
What the G.P. Wants from a Casualty Department. By A General Practitioner	167
Casualty Departments. By Herbert K. Bourns, M.B., F.R.C.S.	168
Jaundice with Hepatic Failure (A Clinico-Pathological Report)	173
That Backbone. By A. Gordon Heron, M.B., Ch.B.	183
Home Care of the Diabetic. By C. T. Andrews, M.D., F.R.C.P.	193
Child Life in Past Times. By S. W. Hinds, M.D., M.R.C.P.	199
The Changing Pattern of Pulmonary Tuberculosis. By R. A. Craig, M.D., B.Sc., M.R.C.P.	206
The General and Special in Medical Practice. By E. M. Bluestone, M.D.	215
Haemorrhage from a Cavernous Haemangioma of the Cord Clinically Resembling an Ascending Myelitis (A Clinico-Pathological Report)	219
Editorial Notes	31, 69, 141
Annotations	20, 100, 130, 225
Correspondence	61, 130
News from Societies	20, 62, 131, 176, 222
Appointments	23, 65, 101, 138, 179, 227
Notes and News	27, 67, 100, 137, 178, 226

Photograph: J. H. BROMHEAD

ERIC WATSON-WILLIAMS

Bristol Medico-Chirurgical Journal

REPORTER 1922
EDITORIAL COMMITTEE 1926
ASSISTANT EDITOR 1933
EDITORIAL SECRETARY 1946
EDITOR 1948–1951

Foreword

BY

SIR PHILIP MORRIS, C.B.E., M.A., LL.D.
Vice-Chancellor of Bristol University

I am glad that I was asked to contribute a foreword to the *Medical Journal of the South-West* because it obliged me to delve into the early issues of *The Bristol Medico-Chirurgical Journal*. I found the quarrying which it was necessary for me to do most interesting and rewarding. The *Medical Journal of the South-West* is, I trust, to be an old friend under a new name, and it will do well to retain the general principles which inspired the policy of its predecessor. There can be few better sources for the discovery of the early history, not only of the Medical School and medicine, but also of the University College and the University. The long period covered by publications of *The Bristol Medico-Chirurgical Journal* saw many changes, some of them as great in their way as the change which has come over the medical services since the National Health Service was instituted. A journal which has already adapted itself to many changes is once more proving its vitality by adjusting itself to the circumstances of the day.

The first number of *The Bristol Medico-Chirurgical Journal* appeared in July 1883, and the *Journal* has maintained a record of uninterrupted publication since then. It had its origin in the volume of *Transactions of the Medico-Chirurgical Society* for the years 1874–78, published in 1878. From 1900 to 1951 the name Watson-Williams was continuously associated with the *Journal*. Dr. P. Watson-Williams became assistant editor in 1900 and editor in 1912. Mr. Eric Watson-Williams joined the Committee in 1926, became assistant editor in 1933 and editor in 1948. No reference, however brief, to the history of this *Journal* would be complete without some acknowledgement of the devoted service which Mr. E. Watson-Williams and his father before him, have given to it.

The first number had the sub-title " A Journal of the Medical Sciences for the West of England and South Wales ", and the Medico-Chirurgical Society has now decided, in effect, that the sub-title should become the title. The reference to South Wales seemed to be inappropriate now in view of the regional organization set up under the National Health Service.

One of the principles underlying the National Health Service was that regions should be established round university centres as focal points. In the case of the South-Western region this was particularly appropriate because the Bristol Medical School was an important part of the nucleus from which the University itself developed, and it is clear from the published records that those who were influential in the Medical School had no little influence in the early formative years of the University. In present circumstances a university must regard itself

as having a much larger parish than its own walls enclose. In medical affairs, if she basic principle of establishing regions round university centres is to be fruitful, all members of the medical profession in the South-Western region must feel themselves to be associated with the region's Medical School. Because the *Medical Journal of the South-West* seeks, as part of its policy, to bring about just such an intimate sense of a unity of interest in the advancement and transmission of medical knowledge, it is assured of a useful future. The Society responsible for the publication of this journal has done its part, and if doctors everywhere in the South-West make this journal their own, interest themselves in it, and above all make contributions to it, there is every reason to expect that the *Medical Journal of the South-West* will be as distinguished in its own day as its great predecessor was before it.

"MARASMUS"

BY

A. V. NEALE, M.D., F.R.C.P., D.P.H.

Professor of Child Health in the University of Bristol.

The history of human progress is well reflected in the changing views of doctors about wasting in infants, which has at different times been called marasmus, infantile atrophy and athrepsia. We have moved on a little from the days of Hieronymus Mercurialis who discusses in his *De Morbis Puerorum* of 1583 the various ways in which "bewitchment" of the child was alleged to occur and notes two points to establish the diagnosis: (*a*) "if the wasting does not yield to any natural remedies or skill or (*b*) if there is no other cause external or internal in the child (or its nurse)". A diagnosis of "idiopathy" was dangerous!

Great attempts were made to stem the tide of infant mortality. There is evidence in the London Bills of Mortality that in 1730–1749 three-quarters of all children born alive died before they reached the age of five. By the end of the eighteenth century about half the children born in London died before age five. In the early history of the Foundling Hospital in London (founded 1745) there were terrible years, the calamity of 1756 being the worst, when fifteen thousand infants were brought in and, as a result of overcrowding, lack of adequate feeding, and the presence of mortal acute infection, only four thousand four hundred "lived to be apprenticed"! A legal measure was later passed to compel the parishes of London to keep their "parish infants" in country cottages where they lived and throve. Some light is thrown upon at least one factor in the scene of starvation by a verse contained in a hymn which was regularly used in the chapel of the Foundling Hospital, London, about 1774:

> The helpless babe, by hunger prest,
> Clings to the famished mother's breast:
> In vain it ev'ry effort tries;
> Life's fountains yield it no supplies.

Dr. Andrew Combe, writing in 1841, asked if this mortality constituted a necessary part of Divine Providence which man could do nothing to modify, or whether it proceeded chiefly from secondary causes, purposely left under our own control and which we may render innocuous by making ourselves acquainted with the infant constitution and its particular different functions.

Undoubtedly an attitude of fatalism governed the outlook for many more years and in 1881 Henoch continued to deplore the high mortality from wasting disease in the first years of life. He noted that of 7,815 infants under the age of one year, treated at the Royal Charité Hospital, Berlin, over a period of ten years, 5,368 (70 per cent.) failed to survive, and many of the deaths were due to profound wasting on admission. Henoch was pleased to note that Nature has assigned the newborn child to the mother's breast and disregard of this principle

in those days was fraught with great dangers. Despite the fact that the physician was often left in " sadness and resignation " he could sometimes hope for success in treating a child even when the fat had steadily disappeared, the skin on the face and the whole body had become flabby and wrinkled and the skin brawny with desquamation. Henoch noted that even at this stage the functions of the alimentary canal, might remain quite normal or almost so; and by suitable nourishment and care " we may still avert the threatened exhaustion and initiate recovery ". In the light of our modern knowledge regarding the diverse causes of marasmus it is a happy thought that the capable nurse could, even in those distressing times, resuscitate a dying infant simply by feeding it!

That there was some accurate knowledge about the subject is illustrated by the story of congenital pyloric stenosis. Dr. Patrick Blair (1717) observed " the child was five months old and was so emaciated that he appeared rather to have decreased since the time of his birth, the whole body not weighing above five pounds . . . the coats of the ventriculus were thick and the pylorus was almost cartilaginous so that no nourishment could have passed into the intestines ". Although Dr. Christopher Weber again described the condition in 1758, and Dr. Michael Underwood in 1784, these were rare occasions and no distinctive causes of " infantile atrophy " were discussed. Time passed on, and L. G. Parsons in the *Goulstonian Lectures* of 1924 on " Wasting Disorders in Early Infancy " observed that the subject appeared to be lacking in general interest and was by many still regarded as threadbare despite the persistence of a high death-rate from these disorders. No doubt, as Parsons said, infantile atrophy was principally a " social disease ", infection being a common factor as was shown by its relative rarity in babies completely breast fed. Vicious circles of metabolic disturbance were recognized and stages of decline were reached in which, paradoxically, the basal metabolism increased with progressive emaciation and dehydration, and in which poor retention of mineral salts and nitrogen would upset the whole physiological balance.

Thirty years ago a great deal of discussion occurred about the possibility of pylorospasm, but while much of it was fruitless it did lead to a more general recognition of true pyloric stenosis, mainly through the work of John Thomson of Edinburgh and Frederick Still of London. This advance in diagnosis and the skill acquired by surgeons in performing the Rammstedt operation of pylorotomy led to a recovery rate of 50 per cent. in 1924. These figures have been very greatly improved since. However, there was at the same time much to confirm the older view of Dr. Charles West that the onset of infantile atrophy was often due to the supply of insufficient and unsuitable food rather than to a primary disorder of digestion and absorption. " Intestinal indigestion " was then, as it is occasionally even now, used as a nebulous label to cover inadequate diagnosis.

Considerable advances have been made in the methods of investigation of cases of marasmus and in their precise diagnosis and it is now only rarely that there is a failure to determine the facts. It must, however, be remembered that underfeeding is still by no means uncommon and that sometimes, even now, children may suffer from it in an extreme degree. Air swallowing and secondary projectile-like vomiting may contribute to the starvation and finally an apathetic state with deprivation anorexia may cause death. My first example is that of a

child who seemed to be on the point of death but who was really suffering from starvation like that seen in countries devastated by war.

D.P. was admitted at the age of ten months in the state depicted in the photograph. Prolonged underfeeding, with intermittent secondary aerophagic colic and vomiting reflected an unusual degree of low-grade mothercraft. The gross marasmus with feeble cry and dehydration, apathy and stupor suggested that death was near. No organic disease could be found and there was a wonderful response to a carefully adjusted dietary. Finally the child became quite normal in appearance and behaviour.

A simple laxity of the cardiac sphincter is frequently the reason for habitual vomiting or easy regurgitation especially during the period of milk feeding. There may be considerable difficulty in maintaining caloric balance. Fortunately thickening of feeds and placing the baby in the inclined position with the head raised is often effective. The early introduction of cereal feeding may cause rapid improvement but, in addition, some tricks of nursing may be needed.

B.K.W. whose birth-weight was 7½ lb. was a hungry, restless and rather dehydrated baby of only 10 lb. at four months. Milk feeds were regurgitated from time to time and there was no gain in weight. Addition of a wheal cereal cured the symptoms and the weight went up rapidly to 13½ lb.

Herniation of the stomach at the diaphragmatic hiatus may quickly produce infantile dysphagia, vomiting which may be severe and protracted or appear in short disabling bouts, and " blood in vomit ". Haematemesis in infancy from cardio-oesophageal ulceration is usually due to a hiatus hernia.

A.D. had vomited from the earliest weeks of life and at the age of two months was only a few ounces above his birth-weight of 7 lb. He was hungry and lively. Persistent underfeeding with cows' milk was suspected, but careful observation showed blood streaks and debris in the vomited acid material. A small hiatus hernia was found. By suitable regulation of feeding and the addition of a cereal and propping the baby up after feeds, this condition subsided (clinically and radiologically) and after some months' careful supervision the growth, body weight and general development were normal.

All sorts of persistent infection will seriously interrupt a baby's progress. Congenital syphilis may, however, occur in a well-nourished baby. Congenital tuberculosis (hepato-pulmonary) may be suspected on circumstantial and clinical evidence. Persistent respiratory sepsis in and following the neonatal period may make a child seriously ill with anorexia, fever, metabolic wasting, secondary alimentary irritability, toxic diarrhoea with fermenting stools and excoriation of the perineal skin.

R.T., one of twins, had pertussis and pneumonia at two months of age. His previous good progress stopped and gradually he drifted to below his birth-weight. The respiratory sepsis, upper zone infection and lung collapse were very troublesome. With good nursing and treatment with aureomycin he recovered slowly and is now normal with a weight of 14 lb.

The irritable irritated baby with eczema is a well-known " waster ". The peculiar difficulties in feeding and nursing may soon set up an intractable duel between mother and baby, and both may break down—the mother in tears and the baby in weight. The condition is common and is full of interest as a social problem, for the psychological strains within the family are often an important contributory cause.

Inherited pancreatic fibro-cystic disease has, unfortunately, become common in paediatric practice. Early failure to thrive and to gain weight in spite of an

apparently normal appetite and food intake are suspicious symptoms. The co-existence of bronchial infection, and foetid greasy stools with a low or absent intestinal trypsin, confirm the diagnosis.

S.D. failed to thrive since normal birth. There was troublesome cough and persistent bronchitis. At four months of age he had only advanced from birth-weight of 7¼ lb. to 8¼ lb., despite the fact that he was persistently hungry and took feeds well. The stools were pale and offensive. His duodenal fluid contained normal bile but a very low tryptic activity. It is noticeable in his photograph that despite the " wasting " (failure to gain) he is alert and is trying heroically to counteract the intestinal indigestion which is due to a lack of the pancreatic enzymes.

Whilst the diagnosis of coeliac disease is less frequently delayed than formerly, an advanced stage of wasting may be seen.

D.C. was normal at birth and weighed 8½ lb. She was not breast fed and her health declined in the later months of infancy and although the detailed history is not clear, observation at twenty-three months indicated severe wasting associated with anorexia, abdominal distension and grave weakness. One large pale offensive stool was passed daily. The diagnosis was clearly coeliac disorder which had drifted on probably since about twelve months of age. Eight grams of fat were excreted in the stools daily. There was normal tryptic activity of the duodenal fluid. Treatment has been very effective and her favourable progress is shown in the photograph.

A long list of detectable causes of wasting disease in infancy could be given. Some are self-evident, such as habitual vomiting; oral and oesophageal thrush infection; upper respiratory sepsis with perhaps degrees of snuffling nasal obstruction; feeding difficulty in cleft palate or in micrognathus; episodes of prolonged anorexia in general diseases, such as acrodynia (pink disease), spastic or mentally deficient infants; recurrent vomiting in chronic subdural haematoma, or some forms of congenital heart disease with cardiomegaly; persistent vomiting in pertussis or even in irritative nasopharyngitis may lead to profound wasting; renal insufficiency associated with congenital abnormalities may eventually present as a general wasting syndrome. Endocrine deficiency disorder may greatly inhibit growth and body weight in infancy and some remarkably interesting examples occur, for instance the pituitary gland may be compressed by a parahypophyseal congenital cyst so producing an infantile form of atrophic Simmond's pituitary cachexia. Inflammation, injuries or neoplasm in the mid-brain may disturb the normal neuropituitary mechanisms and lead to similar effects. The infant cretin, whilst not usually showing severe wasting, may appear to be wasted because of its dry skin and wrinkled appearance.

Metabolic derangements leading to wasting in infancy are essentially early manifestations of tissue enzyme defect. A wasted baby with a very large smooth liver may be so affected because too much of its glucose is being converted and fixed as glycogen (von Gierke syndrome); or a baby may similarly develop hepatomegaly with steatosis. In both these conditions there is an unbalanced one-way traffic of enzyme and chemical exchange. Galactosaemia with galactosuria is a perfect example of metabolic enzyme error: milk feeding is not tolerated in this disorder because the lactose is metabolically toxic and leads to rapid liver enlargement, haemorrhagic tendency and acute wasting. A quick decision to feed the baby on a lactose-free preparation will save life (glucose can be metabolized normally) and later a gradual lactose balance is achieved.

The antidiuretic hormone is of low potency in infancy and body-water balance

(the baby's body is 85 per cent. water) is unstable. Sometimes the infant's renal tubular structure, which is functionally immature, varies in its reaction to anti-diuretic influences, and wasting with polyuria and dehydration may call for special considerations of fluid intake until the renal mechanisms for water conservation are established. Renal tubular re-absorption thresholds may be irregular for phosphate, chloride, glucose, bicarbonate, or sodium: in fact one or more of these substances may be excessively lost in the urine and thus produce, in infancy, clinical syndromes of wasting with anorexia. Simple renal glycosuria causes no upset but it may be accompanied by urinary excretion of amino-acids which sets up a train of general tissue disturbance with wasting and later bone changes (Fanconi Syndrome). " Renal acidosis " due to persistent excess loss of base may be suspected when the urine (in fresh specimens) is consistently alkaline in a " marasmic " baby. A recent advance in these metabolic studies has been the recognition that a baby may show wasting, failure to grow, variable anorexia with periodic vomiting, and a peculiar severe constipation, with no abnormal finding but persistent hypercalcaemia.

T.L., whose birth-weight was 5½ lb., has a normal sibling and his parents are healthy. His progress was such that he reached 7 lb. 11 oz. at ten weeks. After this, however, he moved up slowly and at thirteen months he weighed 12 lb. Severe constipation with hard stools, a capricious appetite and a refusal to tolerate mixed diet was henceforth associated with episodes of vomiting and even over several months there was no gain in weight. He was cheerful and playful, but would not gain an ounce! Yet the only abnormality was persistent (idiopathic) hypercalcaemia—the serum Ca varying between 17 and 18 mg. per cent. We have been unable to lower the Ca after three months without vitamin D, and there is nothing to suggest parathyroid disease. The diagnosis of this case of " wasting " is so far a metabolic mystery.

This case is not unique, several others have come to light since we have tested the serum calcium level in suspect cases.

The term " pseudo-pyloric stenosis " crept into the literature some years ago. Severe projectile vomiting with wasting but no visible gastric peristalsis or palpable pyloric tumour was confusing until it was noted that these babies all had congenital adrenal cortical deficiency (infantile Addison's disease), and strikingly, the female infants so affected showed a pseudo-hermaphrodite appearance.

F.F., a full term female 8-lb. baby, after a week of restful breast-feeding, vomited all feeds and " pumped it up ". Severe wasting was manifest at one month. The external signs of pseudo-hermaphroditism and a great decline of plasma chloride decided the diagnosis of congenital adrenal cortex insufficiency (chiefly a poor production of the mineralocorticoid fraction). Desoxycorticosterone acetate (D.O.C.A.) may quickly remedy the syndrome and, as in this case, stop the vomiting and enable a normal salt balance to be maintained with normal feeding.

In all cases of wasting in infancy the possibility of a structural abnormality which needs surgical treatment must be considered. Examples are oesophageal stenosis (oesophageal atresia must be recognized in the first day of life), oesophageal hiatus herniation, or even the rare condition of circular compression from a double aortic ring embracing the tracheo-oesophageal structure in the superior mediastinum. Diaphragmatic herniation, usually through the left diaphragm, may present as a wasting disorder associated with bouts of obstructive vomiting and severe feeding difficulty. Malrotation of the mid-gut may be recognized early by the special character of the bilious vomiting due to the congenital stricture at or near the duodeno-jejunal zone. Congenital mesenteric

cyst (due to a local duplication of the intestine) may be recognized. Occasionally the infantile form of Hirschsprung's disease with visible colonic peristalsis is observed. Recurrent or intermittent intussusception may be detected. Delayed effects of birth injury and particularly the presence of subdural haematoma causing vomiting and wasting in association with the increased intracranial pressure as indicated by bulging anterior fontanelle and an unusual rate of circumferential head enlargement, demands urgent treatment.

There is no doubt that quick and accurate diagnosis of the cause of wasting in an infant is imperative. A baby's reserves are limited and if nothing is done its condition may deteriorate very rapidly.

I am grateful to my colleagues and to the nursing staff in the Bristol Royal Hospital for Sick Children for their general help in this paper and to the University Department of Clinical Photography for the photographs.

REFERENCES

Hieronymus Mercurialis (Jerome Mercuriale): *De Morbis Puerorum*, Vienna, 1583.
Blair, P.: *Philosoph. Trans. Roy. Soc.*, 1717, **353**, 631.
Weber, C.: *Mors ex spasmo pylori*, Thesis, Univ. of Göttingen, 1758.
Underwood, M.: *Diseases of Children*. London, 1784.
Combe, A.: *Management of Infancy*, Edinburgh, 1841.
West, C.: *Lectures on Diseases of Children*, London, 1848.
Henoch, E.: *Children's Diseases*, Berlin, 1881, and New Sydenham Society, Vol. 125, 1889.
Thomson, J.: *Treatment of Sick Children*, Edinburgh, 1898.
Still, G. F.: *Trans Path. Soc. Lond.*, 1898–9, **50**, 86.
Parsons, L. G.: *Lancet*, 1924, **1**, 793 and 939.

PLATE I

(*a*) Malnutrition. (*b*)

D.P.—Aged 6 months. Weight 6½ lb.

D.P.—Aged 7 months. Weight 7½ lb.

PLATE I

(*c*)

D.P.—Aged 8 months. Weight 9 lb.

(*d*)

D.P.—Aged 10 months. Weight 14 lb.

PLATE II

B.K.W.—Wasting due to simple oesophageal regurgitation.
Aged 6 months.

PLATE III

(*a*) Oesophageal Hiatus Hernia. (*b*)

A.D.—Aged 2 months. Weight 7½ lb. *A.D.*—Aged 19 months. Weight 30 lb.

PLATE IV

(*a*)

(*b*)

Wasting due to persistent chest infection.

R.T.—Aged 4 months. Weight 6 lb.

R.T.—Aged 6 months. Weight 14 lbs.

PLATE V

(*a*) *S.D.*—Note the acute devitalizing effect of the relapsing respiratory infection.

(*b*) *S.D.*—Aged 4 months. Weight 8¼ lb.

(*c*) *S.D.*—Aged 4 months. Weight 8¼ lb.

PLATE VI

(*a*) Coeliac Disease. (*b*)

D.C.—Aged 2½ years. Weight 17 lb. *D.C.*—Aged 3½ years. Weight 31 lb.

PLATE VII

Idiopathic hypercalcaemia.
T.L.—Aged 13 months. Weight 12 lb.

PLATE VIII

(*a*) (*b*)

Pseudohermaphroditism (congenital Addison's disease).

F.F.—Aged 6 weeks.
Weight 6½ lb. Birth weight 8 lb.

F.F.—Aged 4 months.
Weight 9 lb.

UNDERWATER BREATHING*

BY

RONALD WOOLMER, F.F.A.R.C.S.

Surgeon Commander, R.N.V.R.

The pressure of the atmosphere at sea-level is commonly disregarded, because we are perfectly adapted to it, but we must know something about it if we are to understand the effect of departures from it.

The weight of the atmosphere at sea-level exerts a pressure of 15 lb. p.s.i., and it will balance a column of sea-water 33 ft. high, or a column of mercury 30 in. (760 mm.) high. As long as the glottis is open, this pressure is exerted equally on the inside and outside of the chest.

A diver in a non-rigid suit 33 ft. below the surface is exposed to 2 atmospheres pressure, or 30 lb. p.s.i. This is the pressure on the outside of his body, and if his chest is not to collapse the air inside it must be at the same pressure.

At 300 ft. the pressure is 10 atmospheres, and the air supplied to the diver must be at this pressure too. The partial pressure of O_2 to which the pulmonary blood is exposed at 10 atmospheres increases more than tenfold, because the partial pressures of water and CO_2 do not rise, and it approaches 1500 mm.

This pressure pushes so much oxygen into solution in the plasma that the tissues have to draw little or none of the O_2 carried in the haemoglobin. This consequently remains unreduced. This in turn means that there is less base available to combine with CO_2, and the carriage of CO_2 away from the tissues may be seriously interfered with.

Similarly nitrogen is driven into solution in the plasma and tissue fluids in great quantities. So long as the pressure driving it in is maintained this does little physical harm, though it may produce nitrogen narcosis. This is an impairment of cerebral function, similar to alcoholic intoxication, which is frequently experienced by divers. Other inert gases have a similar effect, in proportion to their molecular weights. Xenon has recently been shown to have narcotic properties, and argon, which in air at 10 atmospheres exerts a pressure of about 70 mm. Hg., also contributes to the narcosis. This problem of nitrogen narcosis is a very real one. There is a great variation in susceptibility, but many divers are more or less incapacitated at depths of about 300 ft., where the partial pressure of nitrogen is 6000 mm. of mercury.

The nitrogen is, however, an even greater nuisance during ascent. Tissues vary in the amount of nitrogen they can hold in solution at a given pressure, and in the speed with which they take up that amount. The curve of uptake plotted against time is exponential, so we talk about half saturation times, and the slower saturating tissues (such as ligaments and cartilage) take about 75 min. to absorb half their load. Similarly, if the pressure is rapidly reduced after complete

* Received October, 1952.

saturation has been achieved, it will be 75 min. before they have given off half their excess nitrogen, and several hours before they have parted with it all.

During decompression, the excess nitrogen forms bubbles in the tissues, which may give rise to "bends", by which we mean pain in or near a joint, or to bubbles in the bloodstream.

Not only is the speed of ascent limited by this factor, but also the length of time spent at working depth, because the more nearly the " slower " tissues reach saturation the greater will be the amount of excess nitrogen to be got rid of.

Haldane investigated this problem in 1908. He found that body tissues differed from water in being able to become " supersaturated " with nitrogen. They can hold nitrogen at about twice the tension of the ambient atmosphere without forming bubbles of it; and provided the tension in the ambient atmosphere is not less than half that in the tissues bubbles will not be formed, no matter how quickly the pressure drops. Haldane reasoned that if the total pressure was halved the volume of nitrogen liberated would be the same whether the reduction was from 10 atmospheres to 5, or from 2 to 1. It followed that decompression by stages, halving the total pressure at each stage, would enable the diver to get rid of excess nitrogen far more quickly than if he came up at an even rate.

This led to the system of Stage Decompression—based on Haldane's Decompression Tables—which was a great advance on the older system, and which was adopted throughout the world.

Experience in more recent times, however, has shown that the theory does not quite fit the facts, just as Newton's theory of gravity did not quite fit the facts which improved measuring instruments were able to reveal.

It was found, for instance, that Haldane's Safe Decompression Ratio of 2 was too low for deep dives and too high for shallow ones, and his tables had to be altered.

(The Safe Decompression Ratio is the highest ratio of nitrogen tension inside the body to that outside it at which bubbles do not form.)

Very recently Hempleman, working at the R.N. Physiological Laboratory at Portsmouth, has made a new approach to the problem with brilliant results.

Briefly, he has shown that the amount of excess nitrogen in the tissues varies as $D\sqrt{t}$, where D is the depth of the dive in feet and t the time of exposure in minutes. If this value, in " nitrogen units ", is kept below 500 for the goat, no bends will occur on rapid decompression. Goats are rather more susceptible to bends than humans, but to give an adequate safety margin the figure for humans has been set at 475. Hence a diver can remain at 33 feet for nearly 4 hours and come up without a stop. ($33\sqrt{t} = 475$, $t = 225$ min.); but if he descends to 100 ft. his time below, if he is to come up without a stop, is limited to $22\frac{1}{2}$ min. ($100\sqrt{t} = 475$; $t = 22{\cdot}5$). If he has to stay down for longer than this, stage decompression will have to be used. The depth and duration of each stop on the way up can be calculated from the formula.

New tables based on this simple theory have been worked out, and are now being tested by volunteers in the compression chamber at the Royal Naval Physiological Laboratory in a carefully controlled series of experiments. The results so far are most promising, and there is little doubt that the new scheme will replace Haldane's and will result in better performances.

The threat of bends imposes unwelcome limitations on the diver, and since

they are caused by nitrogen bubbles it would seem that they might be avoided by using another gas than air. If the diver is supplied with compressed oxygen instead of compressed air, bubbles form much less readily and the danger of bends is almost eliminated. It is replaced, however, by a greater danger; that of oxygen poisoning. Oxygen exerting a partial pressure of over 3 atmospheres, which would be attained by breathing the pure gas at 66 ft., or air at over 500 ft., is poisonous. It causes twitching, convulsions, unconsciousness and death, sometimes after quite short exposures.

The nitrogen in air cannot therefore be replaced by oxygen at depths greater than 60 ft., and indeed the oxygen content of ordinary air is too great to permit its use in very deep diving, and a mixture less rich in oxygen has to be used for this. However, oxygen is used to replace air when the diver has ascended to a level where it will not be poisonous. This hastens the elimination of nitrogen, and enables the remainder of the ascent to be completed much more quickly.

The replacement of nitrogen by helium confers certain advantages. Helium is less soluble and more diffusible than nitrogen, so that the tissues attain equilibrium with it more quickly. Similarly, it leaves the tissues faster during decompression. The safe decompression ration is less for helium (about 1·7) than for nitrogen (about 2), and in stage decompression with helium the first stop has therefore to be deeper than with nitrogen. However, helium under increased pressure causes no intoxication, presumably because of its low molecular weight, and herein lies its great advantage over oxygen. It has permitted much greater depths to be attained, and it was on a helium-oxygen mixture that P.O. Bollard with H.M.S. *Reclaim* at Loch Fyne reached the depth of 540 ft. (17½ ats. abs.) in 1948. This depth has not yet been surpassed.

An interesting point about helium is that it is a much better conductor of heat than is nitrogen, and divers using an oxygen-helium mixture have to be provided with electrically-warmed suits to prevent them from getting chilled.

For shallower diving, mixtures of nitrogen and oxygen have been developed at the Admiralty Experimental Diving Unit. These were required in particular for the Mine Recovery Units, which had to be quickly developed for clearing harbours after D-Day. For this work self-contained suits were required, as it was not considered justifiable to risk blowing up more than one man. A lone diver cannot use stage decompression to avoid the bends, so mixtures rich in nitrogen could not be safely used. Similarly, a lone diver dare not risk oxygen poisoning, so he may not use pure oxygen to avoid nitrogen bubbles. A mixture of nitrogen and oxygen, in suitable proportions, though not guaranteeing the avoidance of either, will considerably reduce the risk of both, and the self-contained Mine Recovery Suit, using such a mixture, has been very successful. The fact that all this equipment, down to the last nut and bolt, had to be non-magnetic greatly complicated its manufacture.

The submariner and the frogman have raised many interesting problems in respiratory physiology, and much good work has been done in back rooms to solve them.

A submarine usually carries a complement of about sixty men. It may have to remain submerged for long periods, and the atmosphere must remain respirable. Submarines carry very efficient soda lime canisters for absorbing carbon dioxide, chemical oxygen generators and oxygen compressed in cylinders.

The trend is always towards making the submarine a real undersea boat—one that will remain submerged for a month on end—and this raises difficult storage problems. In a month a submarine's complement would consume nine tons of O_2 and produce eight tons of CO_2, so the magnitude of the problem is apparent. It would be much easier to prevent the CO_2 from rising above a certain level—say 2 per cent.—than to keep it right down to physiological limits. What is the maximum level of CO_2 that can be borne without ill effects for a month? Nobody knows. All previous work has been in the nature of short-term experiment. There is some evidence to show that a few hours' exposure to 2 per cent. CO_2 will result in severe symptoms if the O_2 is at the same time reduced to about 16 per cent. of one atmosphere, but that it causes no symptoms if the O_2 tension is normal. A large-scale experiment, in which a fully-manned submarine will be shut down for a month, is planned to settle this question.

The "snort" or breathing tube introduced to submarines during recent years also brings physiological problems with it. A submarine's diesel engines take in air at a tremendous rate. With the hatches closed, and the ship running below the surface, all this air has to come through the snort tube. But the snort leaves a tell-tale wake, so it has to be kept small, and the required flow of air can only just get through it. Hence there is quite a steep pressure gradient along the tube, and during snorting the air in the submarine is below atmospheric pressure. The pressure is often as low as 600 mm., equivalent to an altitude of nearly 7,000 ft. which would give a partial pressure of oxygen of only 80 mm. in the alveolar air. But if the sea is choppy conditions are more interesting. If a wavelet tries to get into the top of the snort tube, which may be less than a foot above the surface, the float valve automatically closes, and remains closed until the wave has gone by. But the diesels don't stop devouring air, and the pressure falls steeply until the valve reopens, when it regains its previous level. This corresponds to being repeatedly shot up to about 15,000 ft. altitude and down again to 6,000 ft., and the effect can be quite disconcerting, especially on the eardrums. When it becomes possible to do away with internal combustion engines which require atmospheric air, the snort and its attendant problems will disappear.

The most interesting physiological problems, however, are associated with escape from sunken submarines. There are four possible methods. The pressure hull of a submarine, housing the crew, is a rigid tube in which the air is kept at atmospheric pressure. The depth to which a submarine can go is limited by the strength of this tube. At depths greater than a few hundred feet, the hull would be crushed by the pressure of sea-water, and if a submarine is holed and sinks to crushing depth there is no question of escape. Practical methods are therefore concerned only with escapes from up to three or four hundred feet. Three of the four methods involve compression of the crew to the ambient pressure, with subsequent decompression during ascent. In the fourth method, the diving bell, in which a second rigid tube is connected to the pressure hull, the crew remain at atmospheric pressure all the time.

The first method is compartment escape. The pressure hull of a submarine is divided into four or five watertight compartments, each with its escape hatch. After an accident, survivors are collected into one of these compartments (or more, if more than one remains intact). The escape hatch in the top of the compartment is surrounded by a collapsible trunk of rubberized fabric, which can be drawn

down as a vertical tube down to within a few feet of the deck, and rigidly secured. The sea cocks are opened, and water floods into the compartment, compressing the air above it as it rises. If the vessel is lying at a depth of 130 ft., where the pressure is 5 atmospheres, the air in the compartment will be compressed to one-fifth of its volume, and when the water has reached four-fifths of the distance to the top of the compartment, no more will come in. The crew have their heads above water, and they are breathing compressed air. The first man then puts on his under-water breathing set, dives under the rim of the trunk, and comes up inside it. Since the pressures on each side of the escape hatch are now equal, it can be opened, and the man opens it and floats to the surface. The others follow, one by one, at intervals of 15–30 seconds. Flooding the chamber need not take more than a few minutes, and during that time the pressure in the compartment increases from 1 to 5 atmospheres. The ears have to be cleared repeatedly as the pressure comes on.

Sensations during flooding up are peculiar, particularly if the depth is over 100 ft. One feels the water squeezing the legs and body with a vicelike grip, and the clothes are pressed flat and wrinkled against the surface of the body. The eardrums bulge, and quickly become painful if they are not promptly cleared. It soon becomes impossible to whistle, owing to the increased density of the air, and the air current caused by moving the hand close to the head will blow the hair about. No difficulty is felt in moving the heavy air in and out of the lungs when the body is at rest; but it becomes an effort with exercise. The voice has a peculiar bleating quality, and this often causes much hilarity—at any rate in practice dives—among the mildly intoxicated personnel. This intoxication is due, of course, not to an issue of rum, but to high partial pressures of nitrogen. In wartime escapes, however, there is not much hilarity during flooding up, as the water's icy grip climbs up the body. The lights have probably failed, and the air may be already foul after prolonged submergence, and perhaps contaminated with chlorine or smoke. Some of the crew will probably have been drowned, and all shaken by depth-charging. They know that if they get to the surface they may find themselves in an extremely unfriendly environment.

The man in command of the group, after an accident, has to decide when to order the compartment escape to begin, and the decision may be a difficult one. He may wish to delay until surface forces have located the submarine, or until daylight and slack tides. The time he can afford to wait will depend upon the number in the compartment, its size, and how long the submarine had been submerged before the accident. It will also depend on whether the air-purifying system is still workable, and on how many of the survivors have submarine escape sets. Rising CO_2 and falling O_2 levels may force his hand. Once he has decided on escape and the chamber has been flooded, the men waiting to go up are exposed to new dangers. Those without breathing sets are building up their tissue nitrogen and have the bends in store, and their cerebration and survival is imperilled by nitrogen narcosis and CO_2 poisoning. Two per cent. CO_2—quite a usual level in a submerged submarine—will exert the same tension, after flooding up at 130 ft., as 10 per cent. would at normal pressure. At the tension of oxygen which will go with it, it will produce stupidity and unconsciousness rather than respiratory stimulation. Those with breathing sets may avoid the bends, nitrogen narcosis and CO_2 poisoning, but they are faced with oxygen

poisoning, and in any case the duration of a set is only about forty-five minutes. It is therefore imperative, once the chamber has been flooded, to get the men out as quickly as possible.

Compartment escape was the method used in H.M.S. *Truculent*, which was holed while on the surface in the Thames Estuary. There were seventy-nine men aboard. Five, on the bridge, were swept into the water as she sank. Ten were drowned by the flooding of their compartments. The remaining sixty-four managed to close the watertight doors and secured themselves in two compartments. Every one of these sixty-four escaped alive from the submarine. The tragedy was that owing to a series of mischances only ten were picked up alive.

The two other methods of escape under pressure are tower escape, from the conning-tower or gun-tower hatch, and individual chamber escape, from a chamber specially fitted in some submarines. In these methods a small chamber, able to hold one or two men, is repeatedly occupied, flooded up, opened to the sea, evacuated, closed from the sea, drained, restored to atmospheric pressure, opened to the interior, occupied and so on. It is a slow method, but the men waiting to escape remain dry and at normal pressure, which may be an advantage. It was by individual chamber escape that the few survivors of H.M.S. *Thetis* got out.

In the diving-bell method the bell is lowered over the escape hatch of the submarine and fastened on. The pressures are then equalized, the hatch opened, and two, three or four people pass into the bell. The doors are closed, the union broken, and the bell raised to the surface. This process has to be repeated many times. The method is comfortable, in that the survivors remain dry and at normal pressure, but it is slow and can only be applied in favourable circumstances. It was used very successfully with the American submarine *Squalus* in 1939.

An underwater breathing set, though useful, is not essential to escape, and the method of " Free Ascent " is less hazardous than it sounds. It is taught and practised regularly from up to 100 ft., and free ascents have been made from 200 and even 300 ft.

At 100 ft. the air in an escape compartment which has been flooded up will be at 4 atmospheres pressure. The partial pressure of oxygen will therefore be one-fifth of 4 atmospheres, and the effect will be equivalent to breathing 80 per cent. oxygen at sea-level. It is well known that after deep breathing of oxygen-rich mixtures it is possible to hold the breath for a long time. Rates of ascent in sea-water vary from 1 to 4 ft. a second, so an ascent from 100 ft. will usually take less than a minute. There is no difficulty in *holding* one's breath for this time. The difficulty is to get rid of it.

Suppose the lungs contain 5 litres of air at the start of the ascent from 100 ft. At 66 ft. (3 atmospheres) this air will have expanded to 6·7 litres, at 33 ft. to 10 litres, and at the surface to 20 litres. If it is not allowed to escape freely, it will burst the lungs, and the volume to be got rid of per second increases logarithmically during the ascent. In the first third of the journey 1·7 litres must be vented: in the second 2·5, and in the last third 5. Even if the pressure does not rise quickly enough to burst the lungs, it may break through the supporting tissues at the periphery. This may result in interstitial emphysema or massive air embolism after surfacing.

Obeying the natural instinct to hold the breath while under water may therefore have fatal results. This instinct can be conquered by training, and many

thousands of successful free ascents have been made as part of the submariner's normal training. In the U.S. the 100-ft. tank has been in operation for several years. In Britain a 100-ft. tank has recently come into use, and smaller tanks have been working for many years.

These 100-ft. training tanks have airlocks at various levels. An airlock is a blister in the tank wall, big enough to accommodate a man. The lower part is filled with water, the upper two feet or so with air at the same pressure. At the bottom of the tank there is a chamber, similar to an escape compartment of a submarine, by which it is entered.

A number of instructors are stationed in the tank at various depths. These instructors remain below the surface for half an hour or so at a time. When an instructor needs air, he goes into one of the locks, puts his head above water, and breathes compressed air. A few deep breaths of this will last him for several minutes. Instructors become adept at moving about in the water with very little effort. They develop remarkable powers of manoeuvre, and become almost aquatic animals.

The pupil about to make a training ascent goes, with an instructor, into the airlock at the bottom. The outer door is closed and water is admitted, compressing the air in the lock. It rises above the top of the inner door, which is about four feet high. When the pressures are equal the inner door is opened. The pupil takes a few deep breaths, ducks under the water and out through the door into the main tank, and begins his ascent. The instructor for the bottom zone has meanwhile glided down from the 75-ft. lock and is waiting for the pupil as he emerges. He conducts him up through his zone, and hands him on to the next instructor. He then either returns to the bottom for the next pupil, or goes back to the 75-ft. lock for a breath of compressed air. When the organization is going smoothly an instructor may conduct two or three pupils through his zone before he needs to renew his air supply. The whole system is carefully co-ordinated, and controlled from the top by telephones and underwater loud-speakers.

A man who has made a free ascent from 100 ft. is pretty confident when it comes to escaping from a submarine. He knows that if artifice fails him he can depend on his own resources to rise uninjured to the surface.

The development of " human torpedoes ", midget submarines, submersible canoes and " frogmen " and Mine Recovery Units during the war raised many interesting physiological problems, which had to be solved mainly by trial and error. These problems concerned not only underwater breathing, but also protection against extremes of temperature, the design of life-belts and buoyant suits that sustain an unconscious man, problems of underwater vision and illumination, protection against underwater explosions, barotrauma, being intelligible on an underwater telephone while wearing a breathing tube and mouthpiece, the best design for " frogmen's " feet, and so on; and they taxed the ingenuity of many medical officers, physiologists and engineers.

Work on all these problems is still going on.

REFERENCES

Boycott, A. E., Demant, G. C. C. and Haldane, J. S.: *J. Hyg. Camb.*, 1908, **8**, 342. The prevention of compressed-air illness.

Hempleman, H. V.: *Med. Research Council Rep.*, R.N.P., 52/708. A new theoretical basis for the calculation of decompression tables.

MEDICINE AT HOME AND ABROAD

BY

JOHN APLEY, M.D., M.R.C.P.

Consultant Paediatrician, United Bristol Hospitals and United Bath Hospitals

The Briton abroad who demands porridge, bacon, marmalade and tea for breakfast may never realize what he has missed; his loss cannot be expressed merely in terms of croissants, black cherry jam and coffee. Meals are not enjoyed only at the dining-table; there are, indeed, banquets at which the ingredients are ideas, the courses are thoughts, and the waiters are words.

It is not only our own compatriots abroad but, equally, visitors to this country, who tend to measure everything by the hallowed yardstick of their national customs; and, of course, always with the naïve assumption that one's own yard and one's own stick are inviolable. I remember being told, on returning from a visit to some children's hospitals in Barcelona, that we have nothing whatever to learn from Spain, apart from guitar playing and bull-fighting; my informant, of course, has yet to go there to confirm this opinion. It was amusing to set against this the remark of a worthy but equally untravelled Spanish physician who, on the sort of occasion when thoughts are uninhibited and tongues loosened, replied to my gambit on English medicine: " Well, Britain *is* an island; and islanders *are* insular ".

Though I have often tried, I find it impossible to assess the impression which visitors to this country take away from any centre of English medicine; and, incidentally, is it not a little disturbing that so few do come here, except from the Commonwealth countries? But then, what would we like our visitors to remember, in Bristol, for example? Perhaps it is easier to be certain of the things we would like them to forget, or never to have seen. We have, there is no doubt at all, many things to be proud of: but what could visitors think of the beargarden at Dr. X's out-patient clinic; or of Mr. Y's surgical spelaeology in the theatre; or of Z's " research ", which we may know only too well is little more than a form of occupational therapy for the medically sterile? We tend naturally, though perhaps subconsciously, to shepherd foreign lambs. When we flock abroad professionally we too are shepherded: we are led to graze in selected pastures, and we must be mindful not to bleat too dogmatically when we return to our own fold.

With this warning in mind it is, I think, instructive and certainly interesting to try to compare the standard of medicine, and for myself particularly of paediatrics, in our own and foreign countries. My own practice when I go abroad is to ask not only the natives but also other foreign visitors for their views: in this way a wider perspective is obtained, though this " random sampling " is obviously open to criticism, and what I choose to regard as a healthy English impartiality may be called by other names. I have tried this gambit in reverse at home, and

have often been tempted to use a sphygmomanometer, or even a thermometer, on my respected colleagues when they accept it, as generally they do with pugnacious alacrity. In the remarks which follow most of the views expressed are those of doctors discussing countries other than their own; though in some instances, of course with a sense of honest indignation, my own opinions insist upon bursting forth.

Thus, it still surprises me to hear so often that in clinical meeicine, an art on which we modestly pride ourselves, other countries consider themselves our superiors. In fact, there must be scarcely a country in the world whose doctors do not believe sincerely that they themselves are the outstanding exponents of bedside medicine. It is not in these islands alone that discussion is confined to who is *second* best. From what I have gleaned, international opinion would award the palm for clinical ability to the French. With the widespread admiration for French diagnostic acumen there is, however, some deprecation of their scientific approach. Moreover, few doctors outside France approve of the polytherapeutic methods in vogue there, which seem inconsistent with the exquisite precision of thought and expression associated with the French. " Just read the advertisements in La Presse Medicale " I have heard said, justifiably, in scathing Bristolian professorial tones; a remark followed by the characteristically honest admission that those in the B.M.J. are almost as deplorable. Paris, which I recently visited with, if I may use the phrase, a " perambulation of paediatricians ", illustrates the world-wide transition between the old and the new in medicine. In medical buildings, as in medical philosophy, there is much patching of ancient structures. In a few cases there has been complete replacement, conforming with the most modern standards; though whether these will survive as long as their predecessors is doubtful. In France, as in other countries, I have been depressed rather than impressed, on seeing the latest productions, by a uniformity which transcends international boundaries; this applies to new hospitals, units for premature infants, medical papers and even thoughts—just as it does nowadays to tanks and tennis. The loss of national individuality leads inevitably to drabness, but perhaps it is good for us.

For a long time German medicine, with its strict but possibly excessively theoretical discipline, was in the forefront of medical progress, but for many well known reasons it has lost its proud position. Countries like Switzerland still maintain and add lustre to the German tradition, though the Swiss have been criticized because so many of their medical departments are subsidized by pharmacological manufacturers. (Some Professorial Chairs in Switzerland, it has been said, are supported not by the traditional pillars of medicine, but by ampoules of synthetic drugs).

It is widely accepted that the mantle of the Germans has now fallen on the Americans. In many illustrious centres in the U.S.A. the standards of medical practice and research are, I agree wholeheartedly, unsurpassed anywhere in the world, and great men and bold pioneers abound. But it must be admitted that there is often a startling gap between them and the more remote units in that vast and heterogeneous continent—a gap which is also evident in their equally vast and heterogeneous medical journals. Most European doctors who read journals in the English language take North American ones in preference to British, though by no means accepting uncritically all that appears in them. The

terms " mechanistic ", " technological " or " luxury " medicine are frequently applied; and a process of transmutation seems to be essential for countries with an older tradition (or a dollar-gap). It is not only in medicine that finance plays a part in moulding the underlying philosophy: wealthy countries like the U.S.A. and Sweden are indulging increasingly in elaborate equipment, and it is of some concern that this trend may lead to disuse atrophy of clinical craftsmanship and judgment.

Sweden takes a justifiable pride in its abundance of first-class engineers, who join their medical colleagues in perfecting ingenious and complicated apparatus. Though this is undoubtedly helpful in promoting some forms of research it may have the unfortunate effect of making the field workers and general practitioners diffident and not self-reliant. Nevertheless, in Sweden I met outstanding men who have greatly advanced medicine, without the aid of machines and without appearing pedestrian. In Norway, doctors have had to keep pace without anything but the simplest equipment; for Norway is as poor as Sweden is prosperous, and medicine is perforce utilitarian, and attractively simple.

Spain similarly suffers the disadvantages and enjoys the independence of poverty. Despite the intense and very attractive individualism of the Spaniard, Spanish medicine still looks towards Germany, even in the neap tide of the German tradition. There has always been a concentration on theoretical rather than practical instruction in Spanish medical schools, possibly due in part to the marked Arabian influence in Spanish history; but the alert and agile-minded Spanish doctors very quickly remedy this defect in practice. In their acquaintance with world medicine those I met were impressively knowledgeable, though, because of native difficulties, they have little opportunity for waging more than a guerilla type of warfare against disease. Large co-operative enterprises and public health work are comparatively deficient, and doctoring is hindered to some extent by the paucity of nursing organizations which are independent of religious establishments.

In preventive medicine figures may speak more clearly than words, and the accompanying table shows the infant mortality rate and the total death rate in the countries I have discussed.*

	Infant Mortality per 1000 live births	Death Rate per 1000 inhabitants
France	55	13.5
Switzerland	34	10.7
U.S.A.	31	9.7
Canada	43	9.2
Sweden	23	9.9
Norway	—	8.9
Spain	68	11.3
England and Wales	32	11.7

* These figures apply to the year 1949, and are the latest available. They were kindly provided by Dr. S. W. Hinds, Lecturer in Child Health and Social Medicine, Bristol University

It will be kept in mind that these figures reflect social and other factors as well as the standard of medicine. It does, however, appear clear that England is one of the safer places to be born in, though possibly it is not equally commendable for those who value longevity.

Of English medicine it is, I think, widely accepted abroad that one branch is pre-eminent, to wit, anaesthesia—a point of view which will probably surprise some of us here. Tribute is paid to the English gift for clear clinical description, and admiration is expressed for the physiological background of English medical work. I believe that the trend of English paediatrics from the hospital to the home is beginning to make a real impression on foreign observers. Perhaps it impresses them all the more as an example of our adaptability, since the whole tremendous field of social medicine is being cultivated here to some extent because of our lack of money, equipment and new buildings.

The subject of research is a difficult one to assess, partly because both quantity and quality have to be taken into account. However, in medical as in other forms of investigation, I think it may be accepted that English fundamental research is outstanding, even though its practical application may sometimes lag behind. From the little I have learned about general practice abroad I have the impression that the average level here is at least as high as anywhere else, and probably is higher than in most countries.

Let me conclude by quoting two distinguished medical foreigners. The first is the very eminent Dr. A. Blalock, who was in Bristol recently. He said he was glad to see signs that British medicine was, at last, losing its " inferiority complex ". The other I quote anonymously (by request) but verbatim: " As for British medicine, somehow it has never attracted much attention. It is considered to be sound and conservative and commands respect but not worship. Possibly the wider modern interest for medical literature in the English language may subsidiarily (for the primary interest is in American papers) bring about a wider recognition of the excellent qualities of your seasoned yet modern concept of medicine ". Even those who may not entirely hold with this statement will agree that a foreigner who writes English as fluently as this has a claim to our attention.

Should any foreign medical men happen, by some chance, to read this collection of random thoughts they will doubtless accuse me of bias and insularity. I will affirm, nevertheless, that if I myself were a patient I should wish to be tended in this country rather than any other. I trust my colleagues will make a note of this for future reference.

THE ELDERLY CHRONIC SICK

The Bristol Local Medical Committee is to be congratulated on its report on the treatment and care of the elderly chronic sick of Bristol. The members of the sub-committee who drafted the main report were Dr. H. R. Dutton, Professor A. V. Neale and Dr. W. B. Williams. There are similarities between the medical problems of the extremes of life, so it was appropriate that Professor Neale should be chairman. The members of the sub-committee acknowledge their particular indebtedness to Drs. P. C. Joscelyne, W. H. Hayes and A. S. Anderson who have held the office of chairman of the L.M.C. in recent years and to Dr. W. Hughes and Dr. R. C. Wofinden who helped with the preparation of the report. Appendices on different subjects have been contributed by Dr. Hughes, Miss M. E. Burgess and Miss B. L. Robertson.

The report lays stress on the size of the problem and puts forward a plea for more exact inquiry. It is difficult to separate the problems of the aged sick from those of the well because the one class shades imperceptibly into the other and because there is no definite borderline between those problems which are medical and those which are social. The committee, therefore, found it necessary to go outside its strict terms of reference and to consider, to some extent, not only the chronic sick but the "frail ambulant", the acute sick and the housing of old people in general.

The problem is an important one and a growing one, and it is to be hoped that the excellent lead given by the Local Medical Committee will be followed up.

Annotation

GOOD SENSE ABOUT ANTIBIOTICS

"The United Bristol Hospitals have recently produced an excellent booklet which offers a practical guide to the use of Sulphonamides and Antibiotics." So runs a warm-hearted annotation in *The Lancet* (April 25th, 1953).

The merits of this booklet were quickly recognized and the first printings were soon gone.

A second edition has just been printed and copies may be obtained from the Secretary, United Bristol Hospitals, Bristol Royal Infirmary, Bristol 2. Price 1s. 6d.

News from Societies

BRISTOL MEDICO-CHIRURGICAL SOCIETY

The Annual General Meeting was held on October 14th in the Physics Lecture Theatre at the Royal Fort, and was attended by over 135 members and guests.

After the Minutes of the previous meeting had been read and approved, the President, Dr. G. E. F. Sutton, M.C., spoke of the deaths during the last year of Professor Rendle Short, Mr. A. J. Wright, and Dr. W. V. Woods. Members stood for a short time as a token of respect.

The President invested his successor, Mr. R. Ramsay Garden, M.A., M.B., D.O.M.S., who gave his Presidential Address on the subject of " Blindness ", which is to be reported in full in the *Medical Journal of the South West*. A vote of thanks for the Presidential Address was proposed by Mr. Jackman and seconded by Mr. Palin.

The meeting then proceeded to elect the following officers:

President-Elect: Dr. Gordon Heron. *Hon. Secretary:* Dr. Brian McConnell. *Hon. Treasurer:* Dr. Macrae. *Hon. Medical Librarian:* Dr. Gale. *Three Members of Committee:* Dr. J. M. Naish and Messrs. Angell James and H. D. Fairman. *Two Members to serve on University Library Sub-Committee:* Mr. Lucas and Dr. Marwood.

In his Annual Report the Hon. Secretary was able to report a most successful year with a very large increase in membership.

The Hon. Treasurer gave a reassuring account of the finances of the Society.

The Hon. Editor of the *Journal* reported certain difficulties which had been met in the past year—causing delay in the production, but gave promise of a remedy of this before many months.

Meetings are usually held at the Royal Fort on the second Wednesday of each month at 8.15 p.m. The following tentative programme has been arranged for the coming session and exact details of subject will be announced in notices sent out before meetings:

November 11th: Clinical meeting at the Bristol Royal Infirmary.

December 9th: R. V. Cooke, Esq., CH.M., F.R.C.S., on malignant disease of the bowel.

January 13th: G. M. Fitzgibbon, Esq., M.D., F.R.C.S., on a subject connected with plastic surgery.

February 10th: Sir G. R. McRobert, C.I.E., M.D., F.R.C.P., on a subject connected with topical medicine.

March 10th: Dr. Beryl Corner and Dr. A. M. G. Campbell on meningitis.

April 14th: Dr. G. F. Abercrombie on a subject connected with general practice.

May 12th: Ashton Miller, Esq., M.D., F.R.C.S., and J. P. Mitchell, Esq., M.B., F.R.C.S., on a subject connected with urological surgery.

CLINICAL SOCIETY OF BATH

President: Dr. G. D. Kersley.

Hon. Secretary: Dr. D. W. Pugh, D.S.C., " Dunedin Lodge ", Weston Road, Bath (Tel. No. 78366).

Meetings are held at the Royal United Hospital, Bath, on the second Friday of each month with cases at 8 p.m. and discussion at 8.30 p.m.

November: Dr. J. R. O'Neill on **"Generalized Lymphadenopathy".**

PLYMOUTH MEDICAL SOCIETY

President: Mr. C. S. C. Prance.

Hon. Secretaries: Mr. W. B. Waterfall, 3 Hartley Avenue, Plymouth; Dr. J. W. M. Owen, Castlehayes, Plympton.

Future meetings:

November 20th: Dr. P. M. F. Bishop on **" Endocrine Therapy ".**

January 15th, 1954: Mr. A. L. Rowse on **" Elizabethan England and To-day ".**

March 5th, 1954: Sir Russell Brain, P.R.C.P., on **" Cervical Spondylosis ".**

DEVON AND EXETER MEDICO-CHIRURGICAL SOCIETY

A meeting of the Society was held at the Princess Elizabeth Orthopaedic Hospital, Exeter, on April 19th, 1953.

After a brief introduction by Mr. Norman Capener, a number of patients were presented by the surgical staff of the Hospital.

Mr. Durbin showed a patient who had recovered from an almost complete pseudo-paralysis due to calcinosis; the extensive infiltration of all muscles with deposits of calcium had resolved by the extrusion of the material through multiple skin sinuses. This patient had been shown to the Society four years previously when completely

bedridden and unable to feed herself. He also discussed the possibilities of reconstructive surgery in poliomyelitis. Apart from the first, these cases and all others shown were ordinary in-patients of the hospital at the time of the meeting, and they demonstrated the range of conditions under treatment.

Mr. Blundell Jones discussed the treatment of paralytic dislocations of the hip in children, secondary to poliomyelitis or to cerebral palsy. The combination of dislocated hips and deficient musculature made walking impossible; the attainment of stable reductions might allow the deficient muscles to develop sufficient control to enable the child to walk with the aid of appliances. If the condition was recognized early enough, the simple procedure of trochanteric ostectomy would stabilize the hips.

The Registrars, **Mr. Merryweather** and **Mr. Robins,** showed patients with intervertebral disc protusion, rheumatoid arthritis and fracture of the talus. Only a very small proportion of patients in the first group came to operation, the majority responding to conservative treatment. The value of a plaster jacket was emphasized; for all spinal conditions the total applied in the hospital out-patient department averaged thirty a month.

Rheumatoid arthritis needed co-operation between physician and surgeon. In the early stages, pain could be relieved and deformity prevented by splinting joints in the physiological position and controlling their movement. In the late stages operation was sometimes indicated to give stability or to correct deformity.

With severe fracture-dislocations of the talus-the urgent necessity was to relieve tension of the overlying soft tissues which jeopardized the viability of the overlying skin and of the foot as a whole.

The Resident Staff then took part in the demonstration. **Mr. Page** showed children with structural scoliosis. The severe scoliosis, especially if developing after an attack of poliomyelitis in infancy, presented one of the most difficult problems in orthopaedics. Three children were shown, each of whom had recently undergone correction followed by an operation for spinal fusion, when aged about twelve.

In the treatment of osteomyelitis, **Mr. Carrier** stressed the importance of early operation if there were not a rapid response to conservative measures. Antibiotics did not reach a focus of infection when the local blood supply was cut off; with the prevalence of resistant strains it was necessary to obtain the organism in order to determine its sensitivity to the various antibiotics.

Miss Matthews illustrated the conservative treatment of congenital dislocation of the hip by presenting children on abduction frames, in plaster and walking in Denis Browne splints.

After these patients had been shown to the meeting, the Secretary, after thanking Mr. Capener and the members of the staff of the hospital, asked whether there was one principle which the orthopaedic unit at Exeter tried in particular to teach. Mr. Capener thought that eclecticism was most important. For example, at least seven types of operation were commonly performed for osteoarthritis of the hip, the procedure being carefully chosen which would best suit the individual need.

SOUTH SOMERSET MEDICAL CLUB

A meeting of the Club was held at Yeovil on September 17th, 1953. The Club was addressed by Dr. C. de W. Kitcat on " The treatment of the tuberculous patient at home ".

The next meeting of the Club will be held at Yeovil on November 19th, 1953, when the address will be given by Dr. Gardiner-Hill whose subject will be the " Adrenal Gland ".

THE SOUTH WEST REGIONAL PAEDIATRIC CLUB

A meeting was held at Cheltenham on May 16th and 17th, 1953. Cases were presented by Dr. C. P. Donnison and Dr. A. F. D. Darlington. A demonstration of Orthoptic cases was given by Mr. J. G. D. Currie and Mr. R. W. Stephenson, with a discussion on the scope and functions of a Children's Orthoptic Department.

Papers were read by Dr. C. P. Donnison, " Congenital Atresia of the Oesophagus ";

Dr. J. A. Crawford, " Some Psychosomatic Syndromes in Child Guidance Practice "; and Dr. Beryl Corner, " Retrolental Fibroplasis ".

TORQUAY AND DISTRICT MEDICAL SOCIETY

1953

October 8th: Dr. Alice Stewart, F.R.C.P., **" The Day-to-Day Work of a University Department of Social Medicine ".**

Dinner and Dance at the Palace Hotel, Torquay.

November 12th: Sir Zachary Cope, **" The Birth of Modern Scientific Medicine ".**

November 26th: Clinical Discussion, **" Is the Health Visitor really necessary? "**

December 10th: Dr. G. H. Tovey, **" Salt and Water in Clinical Medicine ".**

1954

January 14th: Clinical Meeting in conjunction with S.W. Branch of the British Medical Association.

January 28th: Dr. Margaret Jackson, **" Investigation and Treatment of Subfertile Marriages with special reference to the Post-Coital Test ".**

February 25th: Professor Bruce Perry, **" Rheumatic Heart Disease ".**

March 26th: Dr. F. E. Camps, **" Death at Rillington Place ".**

April 22nd: Dr. Hugh Jolly, **" Common Paediatric Disorders in General Practice ".**

June 3rd: Summer Meeting at St. Loyes College, Exeter.

Appointments

UNIVERSITY OF BRISTOL AND UNITED BRISTOL HOSPITALS

Name	*Appointment*	*Formerly*
ANCILL, R. J., M.B.	Lecturer in Anatomy.	Demonstrator.
BAILY, R. A. J., M.B., F.R.C.S.	Senior Registrar in Orthopaedic Surgery.	Registrar, U.B.H.
BOLTON, F. G., M.B., M.R.C.P.	Senior Registrar in Pathology.	Registrar, U.B.H.
BOURNS, H. K., M.B., F.R.C.S.	Consultant Surgeon in Charge of Casualty Department.	Senior Registrar, Plymouth.
CANTRELL, G. R. M.B., D.O.	Registrar in Ophthalmology.	Royal Eye Hospital, Manchester.
CLUTTON-BROCK, J., M.B., F.F.A.	Consultant Anaesthetist and Lecturer in Anaesthetics.	Consultant Anaesthetist, Birmingham.
COLSON, G. M., B.M., M.R.C.P.	Senior Registrar in Medicine.	Senior Registrar, Exeter (Transfer).
DOWNIE, C. C., M.B.	Assistant Medical Officer, Student Health Service.	S.R.O., Bristol Royal Infirmary.
EDWARDS, E. M., M.B., M.R.C.O.G.	Registrar in Obstetrics and Gynaecology.	Redhill County Hospital.
FREEMAN, R. H., M.B., F.R.C.S.	Registrar in Orthopaedics.	Robert Jones and Agnes Hunt Hospital, Oswestry.
GUY, J., MISS, M.B., D.C.H.	Registrar in Pathology.	Demonstrator in Pathology, Bristol University.
HADFIELD, G. J., M.B., F.R.C.S.	Senior Registrar in Surgery.	Registrar in Surgery, United Bristol Hospitals.
HUDSON, G., B.SC., M.B.	Lecturer in Anatomy.	—
JOHNSON, D. H., M.D.	Registrar in Pathology.	—

Name	*Appointment*	*Formerly*
MACGREGOR, W. G., M.B., F.R.C.S., M.R.C.O.G.	Lecturer in Obstetrics and Gynaecology.	Senior Registrar, Bristol.
MEEK, E. S., M.B.	Research Assistant in Pathology.	—
MOYLE, I. R., M.R.C.S., D.M.R.D.	Registrar in Diagnostic Radiology.	Registrar, Westminster Hospital.
MITCHELL, J. P., M.B., F.R.C.S.	Consultant in Genito-Urinary Surgery.	Senior Registrar, Bristol.
PARTRIDGE, J. P., M.B., F.R.C.S.	Senior Registrar in Surgery.	Registrar, Royal Northern Hospital.
ROSEVERNE, V., M.B., D.M.R.D.	Registrar in Diagnostic Radiology.	Merthyr and Aberdare Group of Hospitals.
ROSS, F. G. M., M.B., F.F.R.	Consultant in Diagnostic Radiology.	Senior Registrar, Post-graduate Medical School.
ROBERTS, J. A. FRASER, D.SC., M.D., F.R.C.P., F.R.C.S.(E).	Consultant in Medical Genetics.	—
RUEBNER, B., M.B.	Registrar in Pathology.	Demonstrator in Pathology, Bristol.
SLEIGH, B. E., MISS, M.B., D.A.	Registrar in Anaesthetics.	Resident Anaesthetist, Bristol Royal Infirmary.
WATSON, P. C., M.B., F.R.C.S.	Senior Registrar in Surgery.	Senior Registrar, Southmead (Transfer).

BRISTOL

Name	*Appointment*	*Formerly*
BARKER, J. C.	Registrar, Chest Diseases.	R.M.O., City Hospital, Gloucester.
CROSS, E. G. W., M.R.C.S., D.P.M.	Assistant Psychiatrist.	Senior Registrar.
FLEMING, A. C.	Assistant Psychiatrist, Hortham.	—
FYFE, A. E., M.B., M.R.C.O.G.	Registrar in Obstetrics.	Registrar, St. Helier Hospital, Carshalton.
GALLA, T., M.B.	Registrar in Psychiatry.	—
GILBERT, P. M., MISS, M.B.	Registrar, Blood Transfusion Service.	Locum, same post.
HARRISON, K. G., MISS, M.B., D.R.C.O.G., D.C.H.	Registrar in Pathology, Frenchay.	S.H.O., Frenchay.
HOFFMAN, J. N., M.B.	Assistant Chest Physician.	Registrar, Bristol.
HULME. A., M.B., F.R.C.S.	Consultant Neuro-Surgeon.	Senior Registrar, Bristol.
JAMES, B., MISS	Registrar in Paediatrics.	—
KNOWLES, R. R., M.B.	Registrar in Psychiatry.	—
LUKIANOWICZ, N., M.D. (LWOW).	Assistant Psychiatrist.	Registrar in Psychiatry.
LINTON, C. D., M.B., D.R.C.O.G.	Registrar in Anaesthetics, Southmead.	—
MANDELBAUM, LISA E. M.B., M.R.C.P.	Senior Registrar, Chest Diseases.	S.H.O. in Medicine, Southmead.
SCHWARTZ, S., M.B., F.R.C.S.	Registrar in Orthopaedics.	Registrar, Manchester.

Name	*Appointment*	*Formerly*
SEARGEANT, P. W., M.B., F.R.C.S.	Registrar in Surgery, Cossham–Frenchay.	Locum Registrar, Cossham–Frenchay.
SIBTHORPE, E. M., M.D., M.R.C.O.G.	Senior Registrar, Obstetrics, Southmead.	Registrar, Old Church Hospital, Romford.
SMITH, K. C. P., M.R.C.S., D.P.M.	Psychiatrist, Child Guidance, Gloucestershire C.C.	—
TROTTER, J. K., M.R.C.S.	Registrar in Anaesthetics, Frenchay.	—
WALKER, W. L., M.B., D.P.M., D.P.H.	Senior Registrar in Mental Deficiency and Child Psychiatry.	Senior Registrar, Moorhaven Hospital, Plymouth.
WELLS, C. E. C., M.B., M.R.C.P.	Registrar in Medicine, Southmead.	S.H.O., Bristol United Hospitals.

NORTH GLOUCESTER

Name	*Appointment*	*Formerly*
ELLIS, R. H., M.D., M.R.C.P.	Consultant in Chest Diseases.	—
FINN, D. P., M.B.	Assistant Psychiatrist.	—
GREEN, D. L. M.B. (SYDNEY), F.R.C.S. (ENGLAND).	Registrar in General Surgery.	—
HART, R. J., M.B.	Registrar, Orthopaedics.	—
HUNTER, R. M. M., M.B., D.P.M., M.R.C.P.	Consultant Psychiatrist.	—
MALLICK, S., B.SC., L.R.C.P. & S., D.R.C.O.G.	Registrar in Obstetrics and Gynaecology.	Registrar in Obstetrics and Gynaecology, Warrington.
TOOMEY, A. G. G., B.SC., M.R.C.S., D.M.R.D.	Senior Registrar in Radiology.	Radiological Registrar, United Bristol Hospitals.

BATH

Name	*Appointment*	*Formerly*
SAVILLE, S., M.B.	Registrar, Anaesthetics.	—
WHELEHAN, J. M., L.R.C.P., & S.I.	Assistant Psychiatrist.	—
YORSTON, R. A., M.B.	Registrar, Surgery of Ear, Nose and Throat.	—

EXETER

Name	*Appointment*	*Formerly*
BOOTHROYD, L. S. A., M.B., F.R.C.S.	Senior Surgical Registrar, Exeter.	—
BRIMBLECOMBE, F. S. W.	Consultant Paediatrician, Exeter.	Consultant Paediatrician, Harold Wood Hospital.
FISCHER, E., PH.D., L.R., C.P.I.	Assistant Psychiatrist, Exeter (Royal Western Counties Institution).	Registrar, Cell Barnes Hospital.
FREEMAN, A. G., M.D., M.R.C.P.	Senior Medical Registrar, Royal Devon and Exeter Hospital.	Assistant Chest Physician Cardiff and District (locum).

Name	*Appointment*	*Formerly*
GURRIER, H. P., M.B., F.R.C.S.	Consultant General Surgery, Exeter (Torquay and Newton Abbot).	Senior Surgical Registrar, Guy's Hospital.
LARGE, J. M., M.B., F.R.C.S.E.	Senior Registrar, Thoracic Unit, Hawkmoor Chest Hospital.	Senior Registrar, Exeter
MINOGUE, F., M.B.	Assistant Psychiatrist (Royal Western Counties Institution).	—
SZUR, Z. L., M.B., D.M.R.D., D.M.R.	Assistant Radiotherapist, Exeter.	—

SOUTH SOMERSET

Name	*Appointment*	*Formerly*
BOULLE, J. R., M.B.	Registrar, General Surgery, Taunton and Somerset Hospital.	Surgical Registrar, Barry Accident and Surgical Hospital.
SCOTT BROWN, J., D.T.M. & H., D.M.R.D.	Consultant, Radiology, South Somerset.	Senior Specialist (Radiology), Colonial Medical Service.

PLYMOUTH

Name	*Appointment*	*Formerly*
ANYAEGBUNAM, P. A. OYE, M.B., D.L.O.	Registrar, Surgery of Ear, Nose and Throat, South Devon and East Cornwall Hospital.	Locum E.N.T. Registrar, Plymouth.
HALL, T. E., M.R.C.S.	Registrar, Psychiatry, Moorhaven.	J.H.M.O., Fulbourne Hospital.
HEWITT, S. R., M.B., M.R.C.O.G.	Senior Registrar, South Devon and East Cornwall Hospital, Plymouth.	Locum Consultant Gynaecologist, Fleet and District, and Yateley and District Hospitals.
PATON, S. L.	Registrar, Mount Gold Orthopaedic Hospital, Plymouth.	—
RITCHIE, E. A., M.B., M.R.C.P.	Registrar, Psychiatry, Moorhaven Hospital.	—
SMITH, J. A., M.B., F.F.A.	Consultant Anaesthetist, Plymouth.	Lecturer in Anaesthetics, Postgraduate Medical School of London.
SMITH, W. H., M.B., D.M.R.D.	Consultant Radiologist, Plymouth.	—

WEST CORNWALL

Name	*Appointment*	*Formerly*
ALLISON, R. C., M.B., D.A.	Assistant Anaesthetist, West Cornwall.	G.P. Anaesthetist, Chesterfield Hospital Group.
HUGHES, W., M.D., M.R.C.P.	Assistant Physician, Diseases of the Chest, West Cornwall (Resident Physician, Tehidy Chest Hospital).	—
MOFFOOT, A. G., MR., M.B., F.R.C.S.	Registrar, General Surgery, Royal Cornwall Infirmary, Truro.	—
WINGFIELD, H. V., M.R.C.S., F.R.S.C.E.	Consultant in Thoracic Surgery, West Cornwall.	—

Notes and News

Professor R. Milnes Walker has been elected to the Council of the Royal College of Surgeons of England and appointed to a Hunterian Professorship. He has also been made a Fellow of University College, London.

Dr. Beryl Corner has been elected F.R.C.P. (London).

Miss C. Rendle Short has been elected F.R.C.O.G.

Mr. G. J. Hadfield has been appointed Arris and Gale Lecturer, Royal College of Surgeons of England.

Dr. E. H. Hare has won the Gaskell Medal and Prize of the Royal Medico-Psychological Association for 1953.

The following have been made Professors at Makerere College, Uganda:

Mr. H. J. Croot (Surgery).

Dr. J. N. P. Davies (Pathology).

Miss Coralie W. Rendle Short (Obstetrics and Gynaecology).

Dr. J. H. Middlemiss has been invited to join the panel of Consultants for the Colonial Medical Service. He visited Africa for three months from March, 1953.

Dr. J. E. Cates has returned from a year in the United States. He held a fellowship at Stanford University, San Francisco, and was working under Dr. J. A. Luetscher on steroids in urine.

Professor Heller led a research team in Uganda, on behalf of the Medical Research Council and the Colonial Office, to investigate " Water Metabolism ". Dr. H. Schnieden was a member of the team.

Dr. J. Gardiner has been awarded a Leverhulme Fellowship and is working with Professor Lennon.

CORONATION HONOURS

O.B.E. (*Civil*): R. A. S. Cory, M.B., CH.B. (BRISTOL).
M.B.E. (*Civil*): Dorothy Delbridge, M.B., CH.B. (BRISTOL) (*née* Staley).

SCHOLARSHIPS AND PRIZES

Bristol Royal Hospital Gold Medal, 1952: Awarded jointly to F. J. Appelbe and N. D. Sanerkin; no award was therefore made of the Silver Medal.
Beaverbrook Fellowship: H. Schnieden.
Markham Skerritt Memorial Prize: Dr. J. Grayson.
Carey Coombs Memorial Prize: Dr. F. G. Bolton.
Francis Dudley Prize: Awarded jointly to B. H. Burns, J. H. Chick and S. J. Strong.
Edward Fawcett Memorial Prize: Awarded jointly to H. G. Handel, Sara Hosegood and Rachel M. Hulbert.
Foyle Prize: Rachel M. Hulbert.
Thomas Francis Edgeworth and Francis Henry Edgeworth Prize: S. C. Jordan.
Richard Clarke Prize in Child Health: F. J. Appelbe.
Mackie Pathology Prize: D. L. Archer.
Hewer Pathology Prize: M. B. Gee and M. L. Cox.
Tibbits Prize: A. J. Webb.
David Rayner Prize in Gynaecology: 1952, M. E. Jackman; 1953, J. P. Bishop.
Gold Medal in Child Health and Paediatrics: Mrs. W. J. Coates.
Crosby Leonard Prize: J. L. James.
Lady Haberfield Scholarship: D. B. Paintin.
Marshall and Clarke Prize: D. B. Paintin.
Russell Cooper Prize: D. B. Paintin.
Sanders' Scholarship: J. D. Jefferies.
Medical Suple Prize: J. D. Jefferies.
Surgical Suple Prize: A. J. Webb.
Henry Clarke Prize: Winifred J. Coates.
Barrett Roue Prize: M. J. Betty.
Augustin Prichard Prize: A. J. Webb.

HIGHER DEGREES AND DIPLOMAS

M.D. (*Brist.*)	D. H. Johnson (with Distinction) R. Maggs R. H. Owen
M.D. (*Birm.*)	R. L. Bishton
M.D. (*Cantab.*)	F. J. Y. Wood
M.Sc.	H. Sosnowick
Ph.D.	E. J. Field
F.R.C.S. (*Eng.*)	R. A. J. Baily Irene D. R. Gregory P. W. Seargeant
M.R.C.P.	Roy Hill J. D. Sheward
M.R.C.O.G.	J. C. S. Leverton
D. P. M. (*Brist.*)	E. G. W. Cross J. D. Orme
D.P.H. (*Brist.*)	M. C. O'Connor G. D. Teague
D.C.H.	Brenda James H. M. Perera June Smith Joan Walker
D.A.	Pamella Hellings

UNIVERSITY OF BRISTOL

M.B., Ch.B. (*Brist.*), *June* 1953

WITH SECOND CLASS HONOURS

Coates, Winifred Jean (with Distinction in Medicine and in Child Health)

Grose, Ruth Janet

PASS

Archer, Desmond Leslie
Bain, Adrian Matheson
Brister, Isabel Kate Elizabeth (with Distinction in Public Health)
Brooks, Thomas Arnold Victor
Cook, John Graham
Coppen, Alec James
Crossley, Rex
Davies, Kenneth David
Downes, Peter William Edward
Edwards, Dorcas
Gee, Michael Barnett
Gordon, Wilfred Edward Lawrence
Jefferies, John Dyment
Lalloo, Ranchor Damodar
MacDonald, Archibald Jeffery
McConnell, Doreen Elizabeth
Nicholas, Philip Owen (with Distinction in Public Health).
Pennefather, Marguerite Eleanor
Perry, James Herbert
Price, Harold
Ree, Michael James
Richards, Thomas David
Shipton, Beryl Maude
Skrine, Ruth Lister
Spry, William Bennett
Stevenson, Audrey Beryl
Taylor, Margaret Gwyneth
Ware, John Walter
Webb, Anthony John

IN GROUP II (COMPLETING THE EXAMINATION)

James, Elizabeth Marjorie

WHERE CAN I LOOK IT UP?

Refresher Courses for General Practitioners.

The articles which appeared in the *British Medical Journal* under the above title from October 1949 to December 1950 have now been published in book form—London: British Medical Association, 1952, 25*s*. A wide variety of everyday medical problems is discussed, and a random choice from the fifty-five topics includes such subjects as the management of the premature baby, measles, the causes and treatment of eczema, delay in the second stage of labour, the failing heart, the management of the epileptic, the treatment of varicose veins, and bronchiectasis. Each chapter is short enough to be read in a few minutes. As a rule aetiology, symptoms, signs and diagnosis are briefly reviewed; the emphasis, however, is on treatment, and detailed instructions are given about established, and a few recent, therapies. Chapters on penicillin, streptomycin and the newer antibiotics describe their mode of action, use and the kind of infection in which they may be effective—information not always readily available elsewhere.

The articles admirably serve the purpose for which they were designed. The book is attractively produced and reasonably priced. It will be of value to practitioners and students, and to those specialists who like to know what is afoot in the department next door.

BRISTOL MARRIAGE AND FAMILY GUIDANCE COUNCIL

Medical practitioners in the South-Western Region may like to be reminded that the Bristol Marriage and Family Guidance Council has a centre at 77 Park Street, Bristol 1. This voluntary body, by private interview, lectures or discussion groups, offers advice to engaged couples on many aspects of marriage and home-making; assists through trained counsellors those whose marriages are in danger of failing; and gives help with all questions connected with parenthood. In four-and-a-half years over fifteen hundred persons have sought its aid. The Council makes no charge for its services but, as the expenses of the Centre are considerable, appeals to those who recognize the merits of its work to become annual subscribers.

BAILBROOK HOUSE · BATH

Tel. Batheaston 8189

FOR the treatment of Voluntary, Temporary and certified patients suffering from Nervous and Mental Disorders.

10 minutes from Western & Midland Region Railway Stations. 2 hours from Paddington.

Terms from 11 guineas a week

Resident Physician

A. GUIRDHAM, M.A., D.M., B.Sc. (Oxon), D.P.M. (London)

MOUNT PLEASANT

and

ST. MARGARET'S

WALTON, CLEVEDON

NURSING HOMES for the MEDICAL TREATMENT of mild nervous and other illnesses, by means of psycho-therapy, E.C.T., occupational therapy, exercises, &c.

Special geriatric care provided.

Terms from £6 6s. a week inclusive

Patients can attend daily for treatment

ELIZABETH CASSON, O.B.E., M.D., D.P.M.

St. Margaret's, Walton, Clevedon, Som.

Telephone: Clevedon 627

Visiting Psychiatrist: GERALD DE M. RUDOLF, M.R.C.P., D.P.M., D.P.H.

Editorial

MORTIMER HOUSE, CLIFTON

This hospital which comes under the Southmead Hospital Group is in anomalous position. It is used by the Group as a maternity hospital, but the building is the property of the Local Authority and is held by the Health Committee. By them it is leased to the Regional Hospital Board. The Bristol Health Committee have now decided that they want Mortimer House back, as they need it for pressing demands on their Mental Health Services. The Regional Board, on the other hand, feel it would be a great pity if about forty maternity beds were lost to the City when already the demand is such on the Southmead Group that patients are being sent out before the end of the normal lying-in period—such is the pressure on the maternity beds. We understand that as the Bristol Health Committee are likely to press for the expiry of the lease of Mortimer House, the Regional Hospital Board are being asked to seek powers from the Minister to build a General Practitioner Maternity Unit in the south of Bristol on land which has been allocated for a new hospital. It is unlikely that a new hospital will be established for a long time to come, but it would be a good idea if a start were made with such a maternity unit. We feel sure it would be a pity if maternity beds were lost in the City without any reasonable expectation of their being replaced in the immediate future.

MISS NOTT OF ST. BRENDA'S

On Wednesday, 14th October, 1953, Miss Nott completed twenty-five years as Matron of this Hospital. St. Brenda's began as a Nursing Home shortly after the First World War, the moving spirits being some of the young consultants returning to practice after the war years. In the 1930s the obstetric side developed at the expense of the surgical and medical, and the Nursing Home was specially favoured by many of the general practitioners in Clifton and Redland. With the coming of the National Health Service it was incorporated in the Southmead Group as a General Practitioner Maternity Hospital.

Throughout her twenty-five years Miss Nott has lived in, and it is not exaggerating to say that she is esteemed by the doctors, held in affection by her staff, and loved by succeeding generations of mothers in Bristol. We congratulate her and thank her on behalf of the profession for the work she has done.

CASUALTY OFFICERS

Some hospitals appear to be having difficulty in recruiting casualty officers. One hospital secretary goes so far as to say that it will not be possible fully to staff a hospital casualty department with newly qualified men in the future, and that some new system will have to be thought out. The position at Cossham Hospital in Bristol became so serious that in recent months the Secretary has had to

circularize the general practitioners inviting them to help in the casualty department on a sessional basis. Sometimes good comes of necessity, and this may be the way that the general practitioners are going to get a foot in the hospitals. We understand there has not been too ready a response from general practitioners; moreover those who have undertaken the work are complaining that some other doctors seem to have a habit of sending to the casualty department much of the work which they should be doing themselves.

Developments will be watched with interest.

NEUROSURGICAL UNIT, FRENCHAY

On 14th October, new theatres and extensions for the department of neurological surgery at Frenchay Hospital, Bristol, were opened by Professor Sir Geoffrey Jefferson, F.R.S., in the presence of about 500 guests. The Lord Mayor of Bristol, Ald. K. A. Brown, presided. Introductory remarks were made by Dr. T. Howard Butler, Chairman of the Management Committee, and by the Lord Mayor.

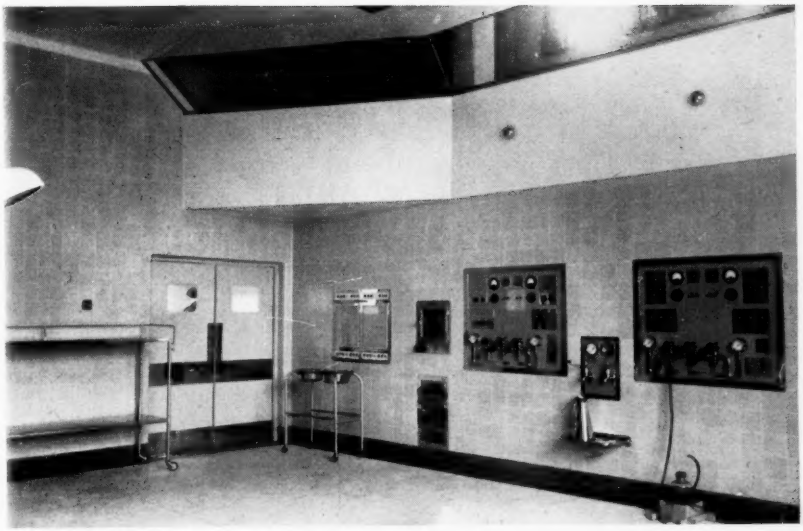
Sir Geoffrey Jefferson remarked that brain surgery was no longer a subject of top-line news in the daily press; he thought the emphasis of public interest had shifted to the thoracic surgeons, with their cardiac surgery. He welcomed this new expansion of the facilities for the neurosurgical service of the Region. He referred to the new premises as a Prospero's dream, but with substance in fact. He described them as probably the finest neurosurgical theatres in existence.

Sir Geoffrey noted with pleasure that Professor Norman Dott from Edinburgh was present, and Miss Beck and Mr. Northfield from London. Mr. Langmaid from Cardiff was also among the guests at this function. After formally declaring the extensions open, and after a benediction by the hospital chaplain, Mr. Greenup, the symbolic opening of the theatres was performed by the cutting of a ribbon with surgical scissors by Sir Geoffrey Jefferson. Mr. G. L. Alexander proposed the vote of thanks to Sir Geoffrey Jefferson. The principal guests then led a tour of inspection of the new buildings, and later tea was served in one of the newly-converted neurosurgical wards.

Among others present were the Lady Mayoress and Lady Jefferson; the Vice-Chancellor of the University of Bristol, Sir Philip Morris, and Lady Morris; Mrs. Norman Dott; the Sheriff's lady, Mrs. Alan Wills; and representatives of the University, the Ministry of Health, the Ministry of Works, and the Regional Hospital Board.

The project was one approved on the appointed day and as such was financed directly by the Ministry of Health. The work was carried out under agency of the Ministry of Works for the most part by local contractors. The layout consists of twin operating theatres and adequate ancillary accommodation. The theatres are air-conditioned and other steps have been taken to minimize aerial contamination when operations are in progress. Substantial improvements to four wards allocated for neurosurgery have been included in the scheme, and a corridor connecting the wards and theatres has been enclosed. The theatres will soon be working [*Plate IX*].

PLATE IX



New Neurosurgical Operating Theatre, Frenchay Hospital.

OBSERVATIONS ON BLINDNESS

The Presidential Address, delivered on Wednesday, 14th October, 1953, at the opening of the Seventy-Fifth Session of the British Medico-Chirurgical Society

BY

R. RAMSAY GARDEN, M.A., M.B., CH.B., D.O.M.S.

Senior Surgeon, Bristol Eye Hospital; Ophthalmic Surgeon, United Bristol Hospitals; Clinical Teacher in Ophthalmology, University of Bristol

" Why was the sight
To such a tender ball as th' eye confin'd?
So obvious and so easie to be quench't "

MILTON: *Samson Agonistes*

When I came to consider possible themes for this address, it seemed to me that some observations on blindness would be an appropriate choice. The subject does not appear to have been discussed at any length in this Society before, and my acquaintance with blind folk of all ages has been an extensive and fairly intimate one, during many years spent in clinical ophthalmology, in certifying the blind in this area, and, until recent years, in looking after the eyes of children at the Royal School for the Blind.

COURAGE OF THE BLIND

I have been further influenced in this choice by a genuine admiration for the courageous and often philosophical attitude of the blind towards their affliction —found not only amongst the aged blind, in whom might be expected a measure of acquiescence in their failing powers. Children born without sight are in a separate category, for they have never known anything different, and in my experience there is a distinctly cheerful, lively atmosphere in a modern school for the blind. The lot of those blinded in youth or in their working years is harder, although they are greatly helped in their adjustment to sightless conditions by a store of visual memories. Nevertheless, whatever the age, previous vocation, or social background of the many hundreds of blind people I have met, I am always amazed and cheered by the spirit in which the great majority of them accept the loss of sight. Possibly those most deprived and handicapped by blindness are the highly educated, intellectually keen elderly people, who have depended for most of their enjoyment in life on reading, and who find difficulty in acquiring fluency in the reading of Braille or Moon type. For them, however, the " Talking Book " has come as a godsend, and with an ever-expanding library

goes far to make up for the deficiencies some people find in broadcasting. Comparative contentment in the blind state may depend on the sympathy engendered in those around; sometimes it may arise partly from a sense of security, for no group of the handicapped have been so well protected by legislation and voluntary effort.

Although some authors have written in the opposite sense, I doubt very much if there is any abnormal psychology of blindness, or any unusual type of personality produced by it. The reaction to blindness is determined, I believe, by the character of the individual, just as in other crises or tragedies in life. The poet Milton, blind from the age of forty-four, is said to have accepted his lot with courage and cheerfulness, and there is little of self-pity in any references he makes to his own blindness—as witness the sonnet addressed to his pupil and friend, Cyriack Skinner:

> Cyriack, this three years day these eys, though clear
> To outward view, of blemish or of spot;
> Bereft of light thir seeing have forgot,
> Nor to thir idle orbs doth sight appear
> Of Sun or Moon or Starre throughout the year,
> Or man or woman. Yet I argue not
> Against heavns hand or will, nor bate a jot
> Of heart or hope; but still bear up and steer
> Right onward. What supports me, dost thou ask?
> The conscience, Friend, to have lost them overply'd
> In libertyes defence, my noble task,
> Of which all Europe talks from side to side.
> This thought might lead me through the world's vain mask
> Content though blind, had I no better guide.

Milton's contentment in blindness, and in the face of his many domestic and other trials, was doubtless strengthened by the literary activities with which his days were crowded.

SOCIAL WELFARE

The literature on blindness in all its aspects is very voluminous, and the social aspects of blindness, the growth of educational and welfare services for the blind, can only be outlined briefly, although their history is absorbing. In most European countries, the record of efforts by religious bodies and philanthropic individuals to relieve the sufferings and poverty of the sightless covers several centuries. Organized help for the blind at first took the form of asylums or hospices, the most famous of which was the Hôtel des Quinze-Vingts in Paris, still in existence today as an ophthalmic hospital and blind institution. This hospice was founded by Louis IX of France about the middle of the thirteenth century as a refuge, it is said, for three hundred of his Crusaders who, captured with him by the Saracens, were over a period of fifteen days blinded at the rate of twenty each day (hence " quinze-vingts ") as a barbaric method of hastening payment of the ransom demanded for the King's release.

Organized education for the blind, however, did not come about until 1784, when the first national school for blind children was established, also in Paris,

by Valentin Haüy. His system of embossing paper with raised characters to provide reading-matter for the blind was later superseded by that of Braille (a pupil and later instructor at the school founded by Haüy), who developed and published in 1829 the six-point embossed type which is now used throughout the world for blind education of all kinds—literary, musical and scientific.

Following the example set in France, Liverpool opened a school for the blind in 1791, Bristol and Edinburgh in 1793, and London in 1799. From these beginnings, a comprehensive system of blind education, welfare and benefits has been evolved in this country, and it is in many respects the most complete in existence. It still incorporates a strong voluntary element—a combination of philanthropy and State financial assistance which has now practically disappeared from so many other national institutions. The activities of the largest of the voluntary bodies—the National Institute for the Blind—include the administration and maintenance of seven Sunshine Home Nursery Schools for blind infants, two secondary grammar schools, a school for blind children with other handicaps, a clinic and institute of physiotherapy, a training school for shorthand and telephony, and two residential rehabilitation centres for newly blinded adults. In addition there is its great publishing department, which prints embossed literature of all kinds (including text-books and scientific treatises for students) for blind persons of all ages.

Bristol's share in the education of blind children, in the training, employment and welfare of blind adults, and in the care of the aged blind, is a long and honourable one. The Royal School for the Blind, the Blind Asylum Workshops, and the three homes for old people, are institutions of which this City can be proud. It may be of interest to recall that the School was founded 160 years ago by two Quakers, Bath and Fox, who started with six pupils in an old meeting-house long since demolished during slum clearance. In 1803, it was moved to larger premises in Maudlin Street, on the site of the present Eye Hospital, and in 1838 to a building near the top of Park Street, where it remained until finally, in 1911, it came to rest in its present fine building in Henleaze Road. (The *Western Daily Press* of 4th February, 1953, contains an interesting account of the School and its development.)

The rehabilitation of the newly blinded is very important. Every year over 2,000 men and women between the ages of twenty-one and sixty-five years are added to the Register in England and Wales. They must not be allowed to brood and rust, but should at once be made to realize that they can become useful citizens again. They are usually in hospital or attending out-patients when the decision to recommend registration is taken, and through the Almoner and the local Blind Association application is made for admission to a Home of Recovery. There they learn to overcome difficulties in performing the usual tasks of daily life, to get about unaided, and gain facility in reading Braille, or in the case of older people the easier Moon type. Lessons in typewriting and handicrafts are given to most of the residents, and later on each individual's capabilities can be assessed. In the workshop, the sense of touch and manual dexterity can be developed by assembling parts, using a lathe, the Braille micrometer and other tools. Later, some may proceed for further training to Government centres, and some may perhaps return to take a useful share in their previous job, or other sighted industry. Two years ago, with a number of colleagues, I

spent a most interesting morning at Manor House, Torquay, one of the Queen Elizabeth Homes of Recovery for the Newly Blind. This house, and America Lodge (the gift of the British War Relief Society of the United States), are maintained by the National Institute for the Blind. There one finds people from all walks of life, spending varying periods of re-adjustment to new conditions, regaining confidence and hope, and eventually returning better fitted physically and mentally to take an active part in the life of the community.

The work of St. Dunstan's in the rehabilitation, training and re-settlement of blinded Servicemen is well known, but mention should be made of its activities and those of the National Institute in the invention and production of devices to help the blind in their work, recreation and locomotion. These range from writing machines, Braille watches and engineers' instruments, embossed playing-cards and games, to metronomes for musicians, and the "talking book" for those (and they are the majority) who have not mastered Braille or Moon type sufficiently to read embossed literature with ease and enjoyment. This invention, which I have seen, is a long-playing electric gramophone, adjusted to run at 24 revolutions per minute for British recordings, with an alternative speed of $33\frac{1}{3}$ r.p.m. so that American records can be played. It is so arranged that the users can do without sighted help, even when it comes to fitting a new pick-up. About 900 book titles are now available, chosen to suit various literary tastes, and are being added to at the rate of about fifty new recordings a year.

In regard to employment, the aim and tendency nowadays is to integrate the adult blind with the sighted as closely as possible. This is reflected in the increasing numbers of the blind who are employed in factories, and in a few new industries which are now within the capabilities of the blind.

INCREASE IN BLINDNESS

Although we may well be satisfied with the progress of social welfare for the blind, there are other aspects which are not so pleasing, and some of them are important enough to deserve more detailed attention than could be given them in the general survey or progress-report I had originally contemplated. To begin with, the rising tide of the registered blind is disturbing, and since the problem of stemming it is by no means entirely an ophthalmological one, I thought this occasion might be an opportunity to place it before an audience comprising so many diverse sections of our profession.

Our methods of ascertainment and certification of the blind, if not yet entirely satisfactory, as will be explained later, are at least the equal of any used abroad. The document in use, familiar to the certifying ophthalmologist as B.D.8, is in process of being modified, mainly in the matter of etiological classification, which it is suggested should in future be left to a board of assessors to whom the clinical findings would be submitted. This would lead to uniformity, and might also be a step towards international agreement on nomenclature.

The definition of blindness in the adult and in the child is different—one being industrial, based on the individual's fitness for any sighted job, the other being wider and concerned with the impossibility of education by sighted methods. Some awkwardness occasionally arises when a child, leaving a school for the blind at the age of sixteen, is not regarded as blind industrially and has to

take his chance, without training, in a sighted labour market. These considerations open up many questions, however, and need not be discussed now. What concerns us at the moment is the number of blind people certified under either definition.

Registration of the adult blind is not compulsory, but the statutory benefits which accrue have in the past ensured that a high percentage of those who come within the definition of blindness do actually apply for registration. A certain proportion of blind people, however, estimated at 10 to 15 per cent., remain outside the register—some because no one has suggested an application, others because the extent to which sight had failed had not been realized, and again because many do not appreciate that the standard of sight allowed by the definition and its interpretation is far higher than the word " blindness " usually connotes. I have even met people with exceedingly poor vision who had an illogical but in a way understandable reluctance to allow themselves to be labelled as blind in any sense. The idea would come to them as a surprise, and seemed the final inescapable blow to their cherished hopes of sight returning one day. On the other hand, I have known many patients with seriously failing sight who began, with great philosophy and courage, to equip themselves for a blind future by learning Braille long before there was any certainty of it being required.

When the Blind Persons Act came into force in 1920, the total of the registered blind was 30,785. It has now reached 86,389 and in the last two years for which figures are available (up to 31st March, 1952) 11,000 additions have been made annually. If we include an estimate of those who would be certifiable as blind but for some reason or another are not registered, the total would probably exceed 90,000. With the addition of another estimate of 10,000 for Scotland, the blind population of this island is around 100,000.

CAUSES OF BLINDNESS

Several very detailed analyses have been published since 1938 based on large numbers of blind certificates completed in the United Kingdom. One of the more recent, by Sorsby (1950), deals with the causes of blindness in England and Wales as shown by certificates for the period 1933 to 1943. (Since my paper was read to the Society, the same author has published a further monograph on " The Causes of Blindness in England ", based on certificates for the years 1948 to 1950. Like its predecessor, it contains many interesting facts, figures and observations, which should be studied by all concerned with the prevention of blindness.)

In 1944, jointly with Professor R. H. Parry, I reported to the Bristol City Council on the 782 cases then on our local Register. Bristol being a city with a population so varied in vocations, and with such a variety of industries, there is within its boundaries a fairly representative cross-section of the English people. Although comparatively small in number, the Blind Register in Bristol (now containing 935 names) forms in a sense a microcosm of the country's blind population.

Between 1944 and the end of last year, i.e. in eight years, 622 new applicants were registered as blind. The individuals in both this and the other Bristol group of 782 previously mentioned were, with few exceptions, examined by

myself, and it is upon experience gained in this connection, and in the course of hospital and private practice, that my personal opinions are based.

It has been reckoned that in about 25 per cent. of the blind population the cause dates from infancy, and in over half of all blind people, the cause of ultimate blindness develops after the age of fifty years. In the Bristol blind population, now numbered at 935, nearly half are over seventy years of age, and the National Statistics for the year to 31st March, 1952 (the last available), show the greatest increase in the 70 + age group. It is therefore obvious that the increasing number of the blind is becoming a geriatric problem of some importance. More people are living to the advanced years when senile changes causing blindness are to be expected, and as the average expectation of life is longer, many who lost their sight in earlier years may survive to increase the numbers of the aged blind.

There is a good deal of evidence to show that towards the end of the eighteenth century the commonest cause of blindness was smallpox. Vaccination and other preventive measures steadily reduced the incidence of this scourge and its associated loss of sight, until by the end of the nineteenth century it had ceased to be important in this respect. As one menace to sight went, another became prominent—in the form of ophthalmia neonatorum. This by the end of the nineteenth and beginning of the present century had become responsible for about 40 per cent. of the admissions to the blind schools of this country. As is very well known, ophthalmia neonatorum has now almost disappeared, and for a baby to become blind in even one eye from this cause has become a great rarity. This has been an outstanding triumph for preventive medicine. Credé's method of treating the eyes of the new-born, compulsory notification of conjunctival discharge within twenty-one days of birth, ante-natal treatment of gonococcal and other infections in the mother, with early and vigorous treatment of the baby's eyes when found to be infected—all these combined to reduce the rate and effects of the disease. With sulphonamides, it became possible to cure a case of ophthalmia in a matter of days instead of weeks and, with penicillin, in a matter of hours.

Thus two of the major causes of blindness have been eradicated, and we have now to consider the present-day situation. The frequency with which one finds cataract, glaucoma, the group of congenital, hereditary and developmental anomalies, and high degrees of myopia, is quite striking. They account for at least half of the total, and although the picture (in Bristol at any rate) is changing in at least one respect—the increasing incidence of blindness due to diabetes—all the surveys studied show a preponderance of the principal causes mentioned.

In the official B.D.8 form, the causes of eye defects leading to blindness are classified in four groups, excluding the one called " No information available ". It will be convenient, I think, to discuss the present causes of blindness within the framework of these four groups, and to follow up with some observations on a few important advances in their prevention and treatment. The categories to be considered are the congenital and undetermined causes, the infectious and bacterial, the traumatic and chemical, and the effects of general disease.

Congenital and Undetermined Causes

This includes conditions such as congenital, hereditary and developmental

defects, myopia, cataract, glaucoma, and retinal detachment. In the first Bristol series, nearly 56 per cent. were included in this category and, in the second, 62 per cent. Although a large volume of research has been going on for many years, we know comparatively little that is entirely reliable about the basic causes of this, the largest group.

There is still much uncertainty about the etiology of the sporadic ocular anomalies present at birth. One often finds instances of normal individuals in families affected by what are generally regarded as hereditary or familial eye diseases, and it has also to be recognized that congenital and other ocular defects caused by intra-uterine toxins may be clinically identical with the hereditary types of anomaly. More will be said on this subject when we come to discuss the second group of causes. We are thus not on very sure ground when it comes to the controversial subject of eugenic control of marriage and parenthood. It has been stated that the effects of its application in reducing blindness from hereditary defects might well be counterbalanced on the whole by the loss of potentially useful citizens.

The ocular defects arising in infancy have been added to in an alarming fashion in recent years by the disease of prematurity known as retrolental fibroplasia, which has already accounted for many cases of blindness in babies. To the intensive research being carried on in various parts of the world on this subject, a notable contribution has been made in regard to its preventive treatment by our own colleagues, C. A. Brown and Beryl Corner (1952).

When we come to consider the eye diseases of uncertain origin in older people, we find that it is not yet known why senile cataract develops, or how one can prevent the structural and other changes associated with that most insidious and treacherous of all eye diseases, glaucoma. Many facts are available about the biochemistry and pathology of cataract—we know what happens, but not much of why it happens. On the other hand, we can restore vision by the old or new methods of cataract extraction, prevent serious loss of sight in many cases of early glaucoma, and preserve what remains in those where deterioration is already considerable.

Cataract and glaucoma are obviously the most common causes of blindness in this country, and when it comes to the possibility of remedial treatment, the patients in whom improvement of sight is feasible are, with a few important exceptions, those with cataract.

Infectious and Bacterial

This group includes the results of venereal disease, tuberculosis, specific fevers, septicaemia and meningitis. The importance of interstitial keratitis and other eye diseases of specific etiology has declined to a noteworthy extent, for some of the reasons which operated in the case of ophthalmia neonatorum, and there has been a steady fall in blindness due to phlyctenular ophthalmia and the infections of childhood. This again is largely to the credit of public health departments which during several generations have done so much, on the one hand to improve the nutrition and hygiene of young children and, on the other, to lessen the risks to sight from infectious diseases by organizing prophylactic inoculations and by maintaining a high standard of treatment in isolation hospitals.

It was mentioned in the last section that intra-uterine influences might determine the occurrence of congenital anomalies, as in toxoplasmosis, for example. Again, Gregg's observation in 1941, that congenital cataract was frequent in babies born of mothers who had contracted rubella in the early months of pregnancy, created great interest at the time, and it is possible that other chorion-passing and placenta-passing viruses, toxins and bacteria may also have similar ill-effects.

On the whole, it appears that the tendency has been for blindness due to infection and bacterial causes to decline, especially in children.

Traumatic and Chemical

Most of the injuries causing bilateral blindness in the relatively small number of cases in Bristol and elsewhere were non-industrial. Much *monocular* blindness is caused through industrial accidents, but for two eyes to be lost or for the second to be blinded by injury at work is not at all common. This, however, does not justify any complacency about safety measures in factories or in the home and streets, especially where children are concerned. I refer, of course, to fireworks, darts, catapults, air-guns and other dangerous toys—the frequent causes of serious injury to the sight of youngsters.

General Diseases

Eyes affected by general diseases are not usually amenable to treatment, and any reduction in this sort of blindness will presumably depend on research into the diseases responsible.

One great disappointment has been in diabetes, about which such high hopes were kindled when insulin was discovered. In spite of the strictest control of diabetes by insulin and associated treatments, retinopathy in young diabetics may show itself in ten to fifteen years, and in older patients within five and ten years after onset of the disease.

At the Oxford Ophthalmological Congress in 1946, Professor Ballantyne ended the Doyne Memorial Lecture on " The State of the Retina in Diabetes " with these words: " The physician has reason to be happy in that having established the diagnosis of diabetes mellitus he can in the vast majority of his cases render his patient 'sugar free' and fit to enjoy his normal activities with an almost normal expectation of life. We must apologize if we disturb his peace of mind, but it is surely important to realize that over 30 per cent. of his patients have, or will acquire, pathological changes in the retina (not to mention cataract and other ocular manifestations of the disease), and that these pathological conditions will persist, and possibly advance to a stage at which they are a menace to vision. Unfortunately the apparently successful treatment of diabetes has no effect whatever in preventing or arresting the march of the retinal disease—the facts are a challenge, especially to ophthalmology, to probe more deeply into the problems of this mysterious disease."

For me at any rate, those words have gained added force during the past few years. An impression that I was seeing more diabetics as out- and in-patients, and that I was certifying more of them as blind, especially the older ones, led me to go into the figures in detail—with the following striking results: In our 1944 Bristol survey, diabetes accounted for only 0·5 per cent. cases of blindness,

in Sorsby's 1950 analysis for 1·8 per cent., but in the Bristol registrations since 1944 the proportion had risen to 5·9 per cent.

In the course of a discussion on " Ocular Complications of Diabetes " held at the British Medical Association Annual Meeting this summer, a number of interesting points emerged. According to G. I. Scott, in those seen within a year of the general disease being diagnosed, the incidence of retinopathy is probably 10 per cent., or less, while in patients who have had diabetes for twenty years or more the incidence is over 70 per cent. Diabetic retinopathy appears to be a distinct entity which can occur independently of hypertension; its incidence increases with the duration of the disease, and is not related to the severity of the diabetes. L. H. Howells, at the same meeting, emphasized that diabetes is becoming more common for a number of reasons—it is a hereditary disease; modern therapy increases fertility, makes childbirth safer, and improves the foetal survival rate in diabetic pregnancies. Moreover, diabetics now have an increased expectation of life, more of them live to develop the various ocular complications, and, as in about 30 per cent. retinopathy of some degree eventually occurs, " its prevention is the most important and challenging problem in the management of diabetes." (The opening papers read at the discussion by these authors have since been published in the *British Journal of Ophthalmology* for December.)

There is no evidence that senile cataract is commoner in diabetics than in non-diabetics of the same age, that senile cataract is accelerated by glycosuria, or that any kind of cataract is inhibited by insulin—or for that matter by any form of medical treatment whatsoever.

The necessity for continuous research in diabetes needs no emphasis, and it is to be hoped that before long the unknown factor concerned in producing ocular lesions, especially retinopathy, will come to light.

ADVANCES IN TREATMENT

In the discussion so far, due credit has been given to State Medicine, and little said of the great part ophthalmology plays in the prevention and cure of blindness. Perhaps that is sufficiently obvious, but it is salutary in a profession like ours to pause now and then, to take a backward glance and re-assure ourselves that true progress is being made, to take stock of our present position and see what remains to be done in the future.

Medical Progress

The efficacy of sulphonamides, penicillin and streptomycin, in certain bacterial eye infections is now well established. The value of the newer antibiotics in those eye diseases known or suspected to arise from virus infections, is at present under critical examination. So far the reports are only moderately encouraging, except in the treatment of trachoma with aureomycin, but it is probably only a matter of time before a useful therapeutic agent is discovered to prevent loss of sight from such viral conditions as dendritic corneal ulcer, herpes ophthalmicus and disciform keratitis.

Of all recent discoveries in the treatment of serious eye disease, ACTH and cortisone easily provide the most dramatic results. The use of these hormones

in the treatment of general diseases will of course be well known to you all, but what the ophthalmologist is so grateful for is something which will be effective in preventing adhesions from blocking the pupil and exudates from clouding the cornea during the course of irido-cyclitis. One used to feel almost helpless at times when treating diseases of the uveal tract, and especially on the occasions, fortunately few, when sympathetic ophthalmia in its own inexorable way was steadily destroying the sight of both eyes. Now they have at least a fighting chance, with this shield to prevent tissue reaction, and to protect delicate structures until the attack of inflammation is past.

Surgical progress

It has been stimulating to work in a period during which so many technical advances have taken place in ophthalmology. In practically every year, including the war years, some new idea in eye surgery has been introduced, and we have travelled far since Daviel in 1747 first successfully removed a cataract, and v. Graefe in 1857 first relieved acute glaucoma by iridectomy. These classical operations are still in use and, although modified in technical details, they have remained important items in the ophthalmic surgeon's repertoire. In 1928, another major event occurred in the history of eye surgery, when Jules Gonin first introduced ignipuncture of the globe to seal off the holes which are usually present in serous detachment of the retina. Then at last it became possible, in suitable cases, to treat successfully a condition hitherto regarded as hopeless. Numerous ideas for replacing detached retinae followed—the principle involved being the use of electrical or chemical applications to the sclera to stimulate adhesions between the choroid coat and the separated area of the retina. By these methods, about 50 to 60 per cent. of all cases of retinal detachment can be cured, and in selected groups the prognosis is much better. In the prevention and treatment of blindness, one is always hoping for some development which will enable one to tackle successfully the type of case which ordinarily does badly, and in the case of detached retinae this has been found recently in the revival of an old idea which has become feasible owing to improved surgical technique. Many cases of detachment do not respond favourably to diathermy operations because for some reason, e.g. shrinkage of the vitreous, the retina does not come near enough to the area of choroidal coagulation to which it is intended to adhere. In such cases, and in others with a bad prognosis (e.g. high myopia, eyes in which the lens has been removed or congenitally dislocated, and detachments in old people), the operation of scleral resection has proved successful in an encouraging proportion of patients. The aim is to shorten the globe, and thus bring choroid nearer retina, by removing a crescentic strip of sclerotic (full thickness, or all but a very thin lamella of scleral fibres) 4 mm. in width at its centre, and 10 to 14 mm. from the corneal margin. Up to half the circumference of the globe may be resected at one operation. The exposed choroid is painted with 1·5 per cent. to 3 per cent. caustic potash to stimulate adhesions to the retina, the sub-retinal fluid is drained off, and very fine silk sutures, previously inserted at the cut edges of the sclera, are tightened and tied to close the wound. A number of unpromising cases of detachment have been treated with good results in Bristol recently, the method used being lamellar sclerectomy, either as a primary operation or following diathermy.

The history of corneal grafting, or keratoplasty, goes back to the early years of last century, but very few successes were recorded in the hundred years which followed. Members of the Society in 1936 had the pleasure of hearing Tudor Thomas, this year's President of the British Medical Association, deliver a lecture on the subject of corneal transplantation, of his own pioneer work on rabbits, and of his successful application of the operation to man in 1930 and subsequently. In the past twenty years, a large number of successful grafting operations have been performed by ophthalmic surgeons throughout the world, and many individual ideas in technique have been devised. Since the passing of the Corneal Grafting Act last year, donor material is becoming available, and corneal transplantation will in future appear more frequently on ophthalmic operation lists than has hitherto been the case. The idea of corneal grafting has made a considerable appeal to the laity, and on more than one occasion I have received touching letters from members of the public, offering one of their own sound eyes to restore the sight of a mother or other near relative. They naturally have little conception as to the limitations of the operation, and expect it to work wonders in all manner of unsuitable eye conditions. Not infrequently, although a clear graft may be obtained, the eye is subsequently found to be unsound in other respects, and the final visual result may not be so good as was hoped. Nevertheless, in a limited number of suitable cases, keratoplasty is likely to be retained as one of our most valuable surgical procedures. It is, of course, possible that there may be fewer opaque corneae requiring surgical treatment in future, owing to the beneficial effects of the sulphonamides, antibiotics and cortisone in reducing the severity of ocular infections.

Cataract surgery has developed considerably in the period under consideration. The older operation, in which the anterior capsule of the lens was opened or partly removed and the hard nucleus expressed, is still in use, although irrigation of the anterior chamber with warm saline is now frequently employed to remove residual cortical debris. The intra-capsular operation, or total extraction of the lens in its capsule, has been steadily gaining favour, however, especially for immature cataract. The technique is more difficult, but the operation has advantages over the other of freedom from post-operative iritis and from any necessity of needling a thickened posterior capsule.

Removal of the crystalline lens, by either method, however, from an eye previously in a normal optical state, renders the eye highly hypermetropic. Before useful sight can be obtained, a heavy and rather unsightly convex spectacle lens is required, in the wearing of which there are some optical disadvantages. The vision is usually clear, but the retinal image is magnified by about one-third, and confusion may also be caused by the prismatic effect of looking through the lens obliquely. Those who have had a cataract removed from one eye, the other having clear natural vision, find they can not see binocularly and may suffer discomfort from the inequality of visual acuity. These difficulties after a successful operation by either of the well-established methods are, of course, minor matters compared with the great benefit gained by cataract patients in recovering useful sight, even although the optical accessories required are rather clumsy. For generations, people have managed reasonably well after cataract extraction by using spectacles, and this is how the majority in future will continue to compensate for the lack of the crystalline lens. The difficulties have been

minimized to some extent and in various ways—for example, by lenses which are lighter, thinner and of special curvature, and by contact lenses. The latter, especially in young patients after a monocular cataract has been removed, have been used with success in a small number of cases, but neat fingers are necessary for their insertion and removal, so that few aged patients are likely to succeed in wearing them.

This brings me to what may prove to be one of the major developments in eye surgery of this century so far. At the Oxford Ophthalmological Congress in 1951, Harold Ridley first described an operation he had devised for replacing the cataractous lens by an acrylic lenticulus of appropriate strength. As a preliminary to the insertion of the plastic lens, the cataract is removed by the older method of capsulectomy, expression of the nucleus, and washing out the residual sticky cortex. Only the posterior portion of the capsule then remains intact, and the acrylic lens is placed in front of that, and behind the iris opposite the pupil, thus restoring the normal optical properties of the eye, except accommodation. At first, the cases selected for operation were middle-aged people with monocular cataract, and the main object was to restore binocular vision. Subsequently, older patients were treated similarly in order to spare them the necessity of wearing heavy lenses for distance, and by now an impressive total of successful results has accumulated. Although the convalescence after the operation is a little prolonged, usually owing to slight reaction to the foreign body by the iris, no serious complications have ensued which could be directly attributable to the presence of the acrylic lens if correctly inserted, by the author's technique, in an eye suitable for the procedure. This means an eye in which the cataract is quite mature, or from which the remaining soft lens matter can be thoroughly washed out after removal of the nucleus. It excludes eyes with very immature cataracts, for which the best procedure is total extraction in the capsule, and some others in which post-operative risks are considerable. Nevertheless, this new operation is a notable advance within its own scope, and the majority of over a score of patients on whom my colleagues and I have by now carried it out at the Eye Hospital are well satisfied with the results.

Glaucoma, on the other hand, remains the most obstinate of eye problems. It is not one condition, but several; it may be acute and congestive, chronic and insidious, it may accompany what is generally considered normal or low intraocular tension, or it may be secondary to other diseases of the eye. The diagnosis may be easy, or so difficult that repeated examinations and provocative tests may still leave the matter in doubt. It is probably true to say that, although intensive research has cast much light on the mechanism, biochemistry and pathology of the various types, no really outstanding development has taken place in the actual treatment of the disease during the time I have been in practice. New miotics have been introduced, and the fashion in some quarters has changed from trephining to iris inclusion operations, but glaucoma in all its varieties is still amongst the most difficult of eye diseases.

THE FUTURE

To conclude this general survey, you might properly expect me to offer some constructive suggestions about ways of reducing the numbers of the blind. There is obviously a need to continue research into the general medical and

ophthalmological diseases which lead to loss of sight. The Institute of Ophthalmology, in the five years since its foundation, has gained an international reputation for advanced work in the biochemistry, biophysics and pathology of the eye. The Director of Research at the Institute is Sir Stewart Duke-Elder, who has himself made such notable contributions to the literature of ophthalmology, and especially to the study of glaucoma. He has on his staff a group of scientists who have already produced an impressive amount of original work. The exceptionally important and recently published communication by Ashton, Ward and Serpell (1953) on " The Role of Oxygen in the Genesis of Retrolental Fibroplasia " is a good example of its high quality. We can take it then that, both here and in centres of research abroad, a wide range of scientific investigation is proceeding. Pending further developments, those of us who are engaged on clinical ophthalmology must concentrate mainly on the treatment of cataract and glaucoma if we are to make any substantial contribution to the reduction of blindness. Sorsby estimated that about 20,000 people in this country were blind from cataract in 1950, and that this number " could be reduced by at least a third if existing methods of treatment were utilized to the full". Of the 11,000 additions made to the National Register, in the last year for which figures are available, I understand that about 3,000 were blind on account of cataract.

As a result of personal experience in examining many applicants for registration, my ideas about improving matters have become to some degree crystallized, and I put forward the following for consideration:

1. The certificate of blindness should be completed by a practising ophthalmologist only, and preferably of consultant rank. Answers are required to such questions as the possibility of treatment and the chance of blindness being cured; it is obvious that an ophthalmologist familiar with the potentialities of modern surgical and medical eye treatment is the only one who can give a reliable and useful opinion. As things are at present, it is quite possible that there are fewer recommendations for cataract and other operations than should be the case.

2. The idea that the patient has to go almost blind, and that the cataract has to be " ripe " before operation can be carried out, is widespread amongst the laity. Actually, the crystalline lens can be removed from the eye of a middle-aged or elderly person by the intra-capsular method, however early the stage of cataract. Operation is justifiable as soon as the eye affected is substantially handicapped by failing vision, and should take place as early as possible, while the patient is still in reasonably good general condition, otherwise the chance of doing it may slip past.

3. Patients' fears about " having to lie in a dark room with both eyes covered up for weeks " should be dispelled, and it should be explained to them that the operation is a brief one, entirely painless, and that both eyes are covered for two days only. The ward or room is shaded, not dark, and the patient is usually up in a week.

4. Out-patients under observation for cataract, glaucoma and other chronic ailments, who fail to keep up their periodical attendances, should be followed up by letter, and a home visit by a Welfare Officer, if necessary. Again and again, I have found at the examination of the blind that they had drifted into a sightless condition through becoming hospital absentees. In some cases it was

due to difficulties about getting to the clinic, in others to apathy or lack of understanding. A Welfare Officer for the Prevention of Blindness was appointed in connection with the City Council's social services in 1945, and she is doing very useful work, not only by securing attendance of patients whose sight is in danger, but also by encouraging them to carry out the advice given in hospital.

5. Glaucoma has been reckoned to be responsible for at least 10,000 cases of blindness in this country; it is much more difficult to deal with than cataract, owing to problems of diagnosis and treatment. One thing certain is that a determined effort should be made to detect the chronic type of the disease in its early stages. Far too many patients first come under the care of an ophthalmologist when gross defects in the fields and even in the central vision have already taken place. It is generally agreed that propaganda addressed to the public on this subject is neither likely to be effective, nor even desirable. The proper approach would be through members of the medical profession who are in contact with large numbers of the population, i.e. general practitioners and industrial health officers. In the United States, the method suggested for screening the population is that of measuring the intra-ocular tension by a simple and inexpensive tonometer, and a crusade is already on foot there, where it is estimated that about one million adults have glaucoma and do not know it. Since the existence of a scotoma near the fixation point is one of the earliest signs of the disease, Williamson-Noble (1953) considers that a simple device for detecting this would be preferable to the tonometer, and he has devised such an instrument. It is hoped that this will be given an extensive trial, for scotoma detection carried out on large numbers of the population might ultimately make a valuable contribution towards the prevention of blindness. Suspected cases would require further investigation, and this could be carried out at special glaucoma clinics attached to the larger eye centres of the clinical areas.

Although several items have been omitted, it is time to bring this balance sheet to a close—on the whole, as I hope will be agreed, a record of substantial progress, with a few disappointments, but a satisfactory reserve of enthusiasm and drive for further efforts. And this will be needed, for it would seem that, so long as the expectation of life increases, and the unexpended portion of intra-uterine life is passed (by the prematurely born) in the supercharged atmosphere of the oxygen tent, those of us who deal with the special senses can see no end to our struggle against the visual and other anomalies which at present inevitably arise. One day, perhaps, when the obstetrician and paediatrician have solved the riddle of prematurity, the physician and gerontologist the problem of reaching old age without senility, we ophthalmologists may be able to write off as settled a few more outstanding accounts.

REFERENCES

Ashton, N., Ward, B., and Serpell, G. (1953). *Brit. J. Ophthal.*, **37**, 513.
Brown, C. A., and Corner, B. (1952). *Ibid.*, **36**, 281.
Sorsby, A. (1950). *Medical Research Council Memorandum* No. 24.
Sorsby, A. (1953). London: H.M. Stationery Office.
Thomas, J. W. T. (1936). *Bristol Medico-Chirurgical Journal*, **53**, 75.
Williamson-Noble, F. A. (1953). *Brit. J. Ophthal.*, **37**, 565.

DIFFUSE ANGIOMATOSIS OF THE SMALL INTESTINE CAUSING MELAENA

BY

JOHN A. VERE NICOLL, F.R.C.S., D.A.

Yeovil

In a recent paper Shepherd (1953) discussed the various types of angiomatous disorders of the gastro-intestinal tract and remarked that few examples had been reported in this country. The following case is therefore interesting, especially because the telangiectasia was more extensive than in previously described patients in whom operation was possible.

CASE REPORT

A married woman, aged thirty-eight and with five children, was urgently admitted to hospital because of extreme tiredness and weakness following a severe melaena on 6th April, 1953. Ten days previously she had returned home after spending fifteen years in Northern Rhodesia. She had felt tired since the birth of her third child six years ago, but all her pregnancies and deliveries had been normal. Her periods were regular, lasting three to four days, fairly heavy, every twenty-five days. Her appetite was fair and she had had no indigestion. She had never suffered from nose-bleeding. There had been severe loss of weight during the past few weeks with increasing weakness and tiredness. There was no history of malaria, dysentery, hookworm infection, or any other serious disease. Recently she had been constipated and had taken "Agarol". Three weeks before admission after taking cascara she first noticed that her motions were very dark.

On her arrival in England ten days before admission, her friends remarked upon her pallor and she noticed it herself. Three days before admission she passed a dead round-worm, and later felt very tired and giddy. The day before admission she felt so weak that she was unable to get out of bed, but she had had no pain.

Family history: Mother alive and well. Father died of carcinoma of the bowel. All her children are well. There was no history of bleeding in any other member of the family.

Condition on examination: A co-operative and intelligent young woman: T.98; P.90 regular; R.24; B.P. 100/60. There was no rash, or enlarged glands. She looked very pale and anaemic. Heart and lungs normal. Breasts normal. Tongue furred—teeth and throat healthy. Abdomen—no abnormality detected on palpitation. P.R.—the rectum was empty and the cervix felt normal. C.N.S.—normal.

Blood picture:	R.B.C.s	= 3·2 millions per c.mm.
	Hb (Haldane)	= 44 per cent. or 6·1 gm. per cent.
	C.I.	= 0·69.
	W.B.C.s	= 11,000 per c.mm.
	Polys.	= 80 per cent. or 8,800 per c.mm..
	Lymphs.	= 19 per cent. or 2,090 per c.mm.
	Monos.	= 1 per cent. or 110 per c.mm.
	Film: R.B.C.s show anisocytosis, poikilocytosis, and hypochromasia.	
	Blood group: "A"; Rh negative.	

No worms, ova or cysts were found in the stools. On 8.4.53 she was given a blood transfusion of four pints. On 10.4.53 her haemoglobin was only 48 per cent. A barium meal

showed no abnormality of the stomach or duodenum. On 12.4.53 she was very weak and giddy when sitting up, and because she was obviously still bleeding a laparotomy was decided upon. The possible diagnoses being considered after ruling out peptic ulcer, were hookworm infestation or Meckel's diverticulum. She was given five pints of blood before, during and after operation, making a total of nine pints.

12.4.53. OPERATION under pentothal, curare, gas and oxygen and ether anaesthesia. A right paramedian incision was made. Immediately, dilated loops of small intestine full of fresh and altered blood were seen. The stomach and duodenum were normal and did not contain blood; but the jejunum presented a most remarkable appearance. Blood-vessels of varying sizes were interlocked and woven in haphazard and tortuous patterns all over the outside of the gut involving at least half of the entire length of the small intestine. The first part of the jejunum was opened, but no obvious internal enlargement of vessels and no hookworms could be seen here or in the duodenum. No Meckel's diverticulum was found and the rest of the bowel and viscera was normal [*Plate X*].

To remove so large a segment of gut in this patient was thought unjustifiable. It was decided to close the abdomen and try to arrest the bleeding by conservative methods, and to continue with blood transfusion. The next day the patient was better and the haemoglobin was 64 per cent. She ate well and did not feel giddy. A full second-stage gastric diet with added vitamins was given. On 16.4.53 the haemoglobin was 52 per cent. and two pints of blood were given. On 18.4.53 the haemoglobin was 66 per cent. and the patient felt well. An enema, however, still produced a melaena stool. The haemoglobin was 64 per cent. on 20.4.53, 60 per cent. on 24.4.53 and 50 per cent. on 25.4.53. A further blood transfusion of two pints was given, but on 26.4.53 the haemoglobin was still only 50 per cent. despite transfusion the previous day, so it was decided to resect the whole affected small intestine. Two pints of blood were given before, one pint during and three pints after the operation.

SECOND OPERATION: The abdomen was opened through the original paramedian scar. Adhesions were present which had to be separated carefully. The length of affected segment and of the whole of the small intestine was then carefully measured, and it was found that about seven feet of intestine had to be removed, leaving about three and a half feet of jejunum proximally and five feet of ileum distally, the whole length being considerably less than the twenty-three feet usually described. After resection and end-to-end anastomosis, the abdomen was closed. On opening the resected segment of gut no enlarged vessels were seen but the mucous membrane had a red blush throughout, and it seemed that there had been steady oozing from all this mucous membrane.

Pathological report:

" Specimen—5 feet of small intestine opened. No macroscopic abnormality noted apart from the blood vessels in the serous coat. These are apparently greatly increased in number and are tortuous. Diffuse telangiectasia of small intestine."

Subsequent progress was rapid and uneventful, and the haemoglobin climbed steadily to normal. Five months after the operation the patient had gained a stone in weight, and was very well. There were no symptoms suggesting impaired fat absorption, and no signs of avitaminosis.

DISCUSSION

It was difficult to decide upon the correct treatment for this patient. At the first operation removal of so large a portion of small intestine was thought inadvisable, especially since spontaneous regression of individual haemorrhagic lesions may occur (Shepherd, 1953). When, however, bleeding persisted, resection was clearly indicated and seemed to offer hope of complete cure, since removal of as much as half the small intestine is not followed by serious metabolic disturbance (Croot, 1952).

PLATE X

Appearance of small intestine seen at operation.

A diagnosis that was considered was hereditary telangiectasia (Osler-Rendu disease). But a meticulous examination of the patient revealed only one capillary telangiectasis over the inner side of one ankle. This had become enlarged and conspicuous during the patient's pregnancies. No other telangiectasis was found. Moreover, while a large proportion of patients with Osler-Rendu disease give a family history of the disorder (Shepherd, 1953), none was obtained in our patient. This case in fact is similar to another recently described as diffuse angiomatosis of the small intestine (Richardson and Flatt, 1953).

REFERENCES

Shepherd, John A. (1953). *Brit. J. Surg.*, **40**, 409.
Croot, H. J. (1952), *Brit. med. J.*, **1**, 195.
Richardson, J. E., and Flatt, A. E. (1953). *Brit. med. J.*, **2**, 311.

PHAEOCHROMOCYTOMA OF ADRENAL GLAND WITH PAROXYSMAL HYPERTENSION.

BY

G. M. COLSON, B.M., M.R.C.P.

Late Senior Medical Registrar, Royal Devon and Exeter Hospital.

Phaeochromocytomata are tumours of chromaffin tissue usually in the adrenal medulla or, less commonly, in sympathetic ganglion tissue. They produce varying proportions of adrenaline and noradrenaline and these pressor substances induce hypertension which may be either paroxysmal or sustained. Although the clinical picture has been clarified in recent years cases are still liable to pass unrecognized, especially if the possibility of the condition is not borne closely in mind. The present report describes a case in which there were typical symptoms for four years before the correct diagnosis was made. It also illustrates some points in management and the dramatic improvement which may follow successful treatment.

CASE REPORT

W. B. a foundry worker aged forty-six, was referred to the out-patient department of the Royal Devon and Exeter Hospital in March, 1952, on account of frontal headache, lethargy, constipation and loss of weight. These symptoms were first noted in 1946 when he was normal on physical examination. In 1948 he described his headaches as severe and occurring in " attacks " of sudden onset whilst dressing, lasting ten to fifteen minutes and associated with profuse sweating. Again on examination he was normal and his blood pressure 120/85. Subsequently all symptoms became more troublesome and by 1952 his weight had fallen by two stones, constipation was severe and his " attacks " of headache more frequent. On mild exertion he was liable to sweat profusely. To direct questioning he admitted that his " attacks " were frequently associated with stooping to lift heavy metal plates at work. He would suddenly develop an uneasy feeling in his chest and neck, an intense throbbing, frontal headache, giddiness, pounding of his heart within the chest and, feeling extremely ill, his face drawn and pale and his body drenched in sweat, he would retire to the lavatory to vomit for five or ten minutes when his attack would pass and he was able to resume his work feeling perfectly well.

On examination he was thin, slightly pale, sweaty and had an anxious facial expression. The cardiac rhythm was regular, rate 84 per minute, and the blood pressure 130/90. The heart was not clinically enlarged, the heart sounds were normal and the lung bases were dry. There were no abnormal masses palpable in the abdomen. The optic discs were normal, the arteries slightly spastic, the veins overfilled, and there were scattered deep haemorrhages and moderate exudates, particularly at the macula.

Towards the end of the examination a spontaneous " attack " was observed. The pulse rate increased to 120 per minute, the heart overacted and the carotid pulsations were strikingly large. The pupils dilated, the facial colour changed to a deathly white and sweat stood out on his forehead. The blood pressure was taken immediately and found to be 290/160. During the next five minutes the blood pressure fell steadily to 125/95 by which time he had regained his normal composure. A clinical diagnosis of phaeochromocytoma was made and the patient admitted for investigation.

During the next three weeks his temperature was frequently raised to 99° F. and the

resting pulse fluctuated between 80 and 100 per minute. The blood pressure, recorded several times daily, remained at 125/95 with the exception of three mornings when the patient complained of headache and the blood pressure rose to 190/140.

The cold pressor response was normal. The response to changes of posture indicated lability of the blood pressure, but postural hypotension and tachycardia were not observed. An intravenous histamine test (Roth and Kvale, 1945) using 0·0125 mg. histamine acid phosphate was positive. From a resting basal blood pressure level of 130/95 there was an immediate fall to 110/80 followed by a dramatic rise in twenty seconds to 300/200 accompanied by the signs and symptoms identical with those of a spontaneous attack. Within seven minutes the blood pressure had returned to 125/90.

The blood count was normal. The blood urea was 58 mg. per 100 ml. The urea clearance (Van Slyke) was normal. Examination of the urine showed a light cloud of protein and the presence of a few hyaline and granular casts. The fasting blood sugar was normal. The heart was normal on screening. An X-ray of the abdomen showed no abnormal calcification. An intravenous pyelogram revealed considerable downward displacement of the left kidney with acute angulation of the ureter at the pelvi-ureteral junction: the right side was normal. An electrocardiogram showed the pattern of left ventricular strain.

A confident diagnosis of phaeochromocytoma of the left adrenal gland was made and on 23rd April operation was performed by Mr. K. P. S. Caldwell. Dr. J. Powell induced anaesthesia with pentothal sodium 0·6 g., followed by nitrous oxide, trichlorethylene and a minimal amount of ether, and continued by the endotracheal route. The patient having been placed in position, tubocurarine chloride 15 mg. was injected intravenously and a further 5 mg. administered subsequently. Through a left lumbar incision a tumour approximately 5 in. × 4 in. was found in the left adrenal gland and removed. The blood pressure remained steady at 120/80 until the tumour was manipulated but then rose suddenly to 280 systolic and finally became unrecordable whilst the vascular pedicle of the tumour was ligated. An intravenous injection of 18 mg. piperidyl-methyl-benzodioxane (933F) was given without any demonstrable reduction of blood pressure. No attempt was made to explore the right adrenal gland in view of the alarming hypertension. On return to the ward the patient's blood pressure fell rapidly to an unrecordable level, he was cyanosed, sweating and his pulse was of small volume. An intravenous infusion of l-noradrenaline (7 micrograms per min.) was effective in restoring the blood pressure to 100/70 and was continued for twenty-four hours when its withdrawal was followed by no fall of pressure.

The subsequent course was uneventful. The blood pressure rose to 130/90 and remained at this level. Protein and casts disappeared from the urine, the blood urea returned to normal and before the patient left hospital the intravenous histamine test was repeated and was negative. When seen in November, 1952, the patient was symptomless and had regained his normal weight. Since operation his bowels had acted regularly each day. On examination he was a fit-looking man with no abnormal signs, the blood pressure being 130/90 and the optic fundi normal. The electrocardiogram had reverted to a normal pattern.

The tumour was oval in shape measuring 12·5 cm. by 10 cm. by 6 cm. and weighed 680 grams. The histological appearances were typical of phaeochromocytoma (Dr. G. Stewart Smith). Biological assay (Dr. W. S. Peart) indicated that the tumour contained 1800 mg. of adrenaline and 1220 mg. of noradrenaline. A sample of urine collected for fourteen hours before operation showed an excretion of 600 micrograms of amine and the ratio of noradrenaline to adrenaline was 0·5 (Case G of Hamilton *et al.*, 1953).

DISCUSSION

This case illustrates the features typical of hypertensive episodes referable to an underlying phaeochromocytoma which, in retrospect, must have been present for at least four years. Sudden attacks of short-lived over-anxiety, headache, palpitation, facial pallor, sweating, nausea and vomiting should always arouse suspicion. Additional symptoms include shortness of breath,

numbness, tingling and coldness of the extremities and epigastric distress which may radiate into the praecordium or neck. In about half the cases there is an aura which is recognizable but difficult to describe (Calkins *et al.*, 1951). Careful interrogation will reveal a history of liability to excessive sweating between attacks in 90 per cent. of patients (Smithwick *et al.*, 1950). Constipation was a prominent symptom in the case described and it is noteworthy that it disappeared immediately after operation. This is an unusual feature which has not been recorded by other observers.

Physical examination between paroxysms is frequently negative but abnormal signs which may be encountered include an unduly moist skin, tremor, tachycardia, pupillary dilatation, weight loss, facial pallor, cardiac enlargement, retinopathy and a palpable abdominal mass. The temperature is commonly elevated to 99–100° F., traces of protein and sugar may be present constantly in the urine and showers of hyaline casts intermittently (Pincoffs, 1929.)

The case described possessed so many of these classical features that the clinical diagnosis presented no difficulty. Unfortunately not all cases are equally straightforward and may be confused with anxiety state, thyrotoxicosis, diabetes mellitus, chronic nephritis, migraine and cerebral tumour (MacKeith, 1944). Of these differential diagnoses the most important is anxiety state in emotionally labile subjects, and unless a paroxysm is observed the distinction may be extremely difficult. It is in these cases that every effort should be made to overcome such difficulty by day-to-day observation and further investigation.

An X-ray of the abdomen may reveal patchy calcification at the site of a tumour if of sufficiently long standing. Intravenous pyelography is normal if the tumour is small but downward displacement of the kidney may be demonstrated if a larger tumour arises from the adrenal gland. Presacral oxygen insufflation is theoretically more likely to be of greater value in outlining smaller tumours but in practice often fails to do so—as in the case recently reported by Ward *et al.* (1953), where the right adrenal outline was considered normal but at operation a phaeochromocytoma one inch in diameter was discovered.

Engel and Euler (1950) stressed the diagnostic value of an increased urinary output of adrenaline and noradrenaline in phaeochromocytoma and whenever there is doubt about the diagnosis a 24-hour sample of urine should be assayed. The normal 24-hour output of amine, 20–70 μg. will be exceeded when a phaeochromocytoma is present, and may reach extremely high levels. Hamilton *et al.* (1953) consider that the estimation of urinary adrenaline and noradrenaline offers the best available special method of diagnosis and point out that no false positive result has yet been obtained.

Of the tests designed to stimulate the release of pressor substances from the tumour so that an attack may be observed, probably the most reliable is the intravenous histamine test introduced in 1945 by Roth and Kvale. As Richardson (1951) points out, its use should be restricted to those cases with normal pressures when the intermittent nature of the symptoms has led to the suspicion that paroxysmal hypertension is their cause. The test is not without risk and a hypotensive blocking agent should be at hand whenever it is performed.

The diagnostic use of antagonistic drugs such as piperoxane or regitin (C7337) is, of course, confined to cases with sustained hypertension and affords no help in those where the blood pressure is normal between paroxysms.

It is not proposed to discuss the details of the surgical approach or the factors governing the choice of anaesthetic which have been carefully considered by Ward *et al.* (1953). In the present case the lack of response to benzodioxane and the immensity of the tumour are of interest and probably not unrelated. In so far as benzodioxane exerts its action in competing with adrenergic pressor substances it is not surprising that a dose of 18 mg. failed to compete successfully with the high blood concentration of pressor substances which must have been attained at the height of operative manipulation. No doubt a larger dose might have counteracted the alarming hypertension during operation but only at the expense of greater postoperative hypotensive collapse.

SUMMARY

A case of phaeochromocytoma in a man aged forty-six with symptoms from paroxysmal hypertension is described. The tumour, which weighed 680 grams, was successfully removed at operation.

My thanks are due to Dr. Charles Seward for permission to publish this case and to those mentioned in the text who were concerned in its management.

REFERENCES

Calkins, E., Dana, G. W., and Howard, J. E. (1951). *J. Amer. med. Ass.*, **145**, 880.
Engel, A., and Euler, U. S. von (1950). *Lancet*, **2**, 387.
Hamilton, M., Litchfield, J. W., Peart, W. S., and Sowry, G. S. C. (1953). *Brit. Heart. J.*, **15**, 241.
MacKeith, R. (1944). *Brit. Heart. J.*, **6**, 1.
Pincoffs, M. C. (1929). *Tr. Ass. Amer. Physicians*, **44**, 295.
Richardson, J. S. (1951). *Practitioner*, **166**, 556.
Roth, G. M., and Kvale, W. F. (1945). *J. Lab. clin. Med.*, **30**, 366.
Smithwick, R. H., Greer, W. E. R., Robertson, C. W., and Wilkins, R. W. (1950). *New. Engl. J. Med.*, **242**, 252.
Ward, G. E. S., Riches, E. W., and Johnson, B. (1952). *Arch. Middlesex Hosp.*, **2**, 133.

HERPES ZOSTER ENCEPHALITIS
in association with the Ramsay Hunt syndrome

Report of a case

BY

R. D. G. CREERY, M.D., M.R.C.P., D.C.H.

Principal Paediatric Registrar, Royal Belfast Hospital for Sick Children; late Medical Registrar, Southmead Hospital, Bristol.

Encephalitis is one of the rarer complications of herpes zoster. The clinical picture of the encephalitis may be fulminating with a fatal outcome, or it may be of a much milder nature with ultimate recovery, as in the case here recorded. The present case is reported as an example of its association with another occasional manifestation, the Ramsay Hunt syndrome, or so-called " geniculate herpes " (Hunt, 1907). This syndrome in its most severe form, comprises peripheral facial palsy, herpetic eruption on the ear, face or neck, and involvment of the auditory nerve.

CASE REPORT

A carpenter, aged forty years, was admitted to hospital on 25th May, 1951. Several weeks before, he had been in contact with a case of " shingles ". One week before admission, he complained of pain in the right ear. Two days later he had difficulty with speech and noticed right-sided facial weakness. Later he became " dizzy " and complained of severe headache and photophobia. On the day before admission his right ear began to discharge, and he felt drowsy. Examination showed an apprehensive man, fully conscious, co-operative and apyrexial. The relevant findings were as follows: complete right facial paralysis (lower motor neurone in type), with loss of taste on the anterior part of the tongue; right-sided deafness (middle-ear in type, as judged by tuning-fork tests). Cold caloric tests were normal. The right ear showed an external otitis with herpetic vesicles in the external auditory meatus and on the pinna. The tympanic membrane was involved, but was not bulging, nor was there any perforation. When standing, with eyes open or closed, he tended to fall to the right. There was no other clinical abnormality. Two days after admission he suddenly became rigid, and lost consciousness for several minutes. He rapidly recovered, but remained drowsy for some hours. A transient weakness of the right external rectus muscle was noted. The plantar reflexes were equivocal, later becoming flexor. Lumbar puncture was performed. The pressure was 260 mm. of cerebrospinal fluid and the fluid was faintly cloudy. The cerebrospinal fluid contained 100 leucocytes c.mm., the majority being polymorphonuclears; protein 130 mgm. per cent.; chlorides 730 mgm. per cent.; sugar 54 mgm. per cent. There were no organisms and the fluid was sterile on culture. He was seen the following day by Dr. A. M. G. Campbell, who felt that this was a case of herpes zoster encephalitis, presenting as the Ramsay Hunt syndrome. From then on, the patient's condition gradually improved, the facial weakness decreased, the herpetic lesions healed, and he was discharged on 19th June, 1951. When last seen as an out-patient three months after admission, he was well and had only a slight facial weakness and a minor degree of deafness.

DISCUSSION

The association of encephalitis with zoster infection was discussed by Schiff and Brain (1930), who reviewed the literature, and recorded a further fatal case.

They concluded that, although in some patients the zoster was a complication of an independent encephalitis, in others an extension of the zoster infection had produced the encephalitic picture. In their case this latter conclusion was supported by the results of cerebrospinal fluid complement-fixation tests. Biggart and Fisher (1938), in their report of a fatal case of meningo-encephalitis complicating herpes zoster, gave a very full account of the clinical and pathological findings. The patient became drowsy five weeks after the onset of herpes zoster in the distribution of the right ilio-inguinal nerve, and later became comatose and delirious. The cerebrospinal fluid contained 20 cells per cmm. Pathologically, there were dorsal ganglionitis, mononuclear infiltration of the subarachnoid space, posterior myelitis, and encephalitic changes such as peri-vascular round-cell infiltration. They concluded that these findings supported the possibility that the encephalitis was due to zoster infection, as the lesions in the medulla and mid-brain appeared to be merely an upward extension of the same lesions found in the cord. The encephalitic picture in the present case comprised drowsiness and headache, followed by a seizure with transient coma and sixth-nerve paresis. The cerebrospinal fluid pressure was high, as in Schiff and Brain's case. An interesting feature was the high proportion of polymorphonuclears in the fluid which probably indicated an early stage of the reaction; mononuclear cells are stated to be more commonly found (Brain, 1951). Unfortunately no virus studies were undertaken in this case. Denny-Brown *et al.* (1944) cast considerable doubt on the concept of geniculate ganglionitis as the cause of the Ramsay Hunt syndrome. In a fatal case which had presented this syndrome, they were unable to show any changes within the geniculate ganglion. They found, however, a definite motor neuritis of the facial nerve. On the other hand, Parkinson (1948) agreed that the primary lesion is in the geniculate ganglion. Critchley (1947) surveyed the present rather confused position of this syndrome, and concluded that until a better term is forthcoming, based upon exact knowledge of the morbid anatomy, the " Ramsay Hunt syndrome " may conveniently be used to describe all cases where facial paralysis is associated with an outbreack of herpes zoster vesicles anywhere upon the head and neck. It is probable that the syndrome cannot be explained on any one pathological lesion, but that it is a further example of the pleomorphism which characterizes zoster infection: at one time there may be a ganglionitis, at another encephalitis, or again, a motor neuritis, or, as is probable in the present case, a combination of all three. The local eccentricities in the features of the syndrome could be explained by postulating a varying attack on the nerves and their ganglia supplying the region of the head and neck. It is suggested that the term " geniculate herpes " might be dropped, as it does not satisfactorily explain the variations in the clinical picture and has little, if any, pathological foundation. Recent advances in the use of antibiotics have produced hopes that some of these drugs might be helpful in the treatment of herpes zoster. Binder and Stubbs (1949), and Finland *et al.* (1949), reported good results with aureomycin. It is to be noted that neither series was controlled. More recent work, in which controls were used, by Carter (1951), and Kass *et al.* (1952), suggests that there is little evidence in favour of a specific effect against the zoster virus by either aureomycin or chloramphenicol. Carter did suggest, however, that secondary infection was avoided, but Kass *et al.* were unable to find any significant difference

between treated and untreated cases. That the treatment of zoster infection with antibiotics is not satisfactory is suggested by the experience of Foster and Jackson (1951) who reported the occurrence of herpes zoster encephalitis in a patient treated with aureomycin for the original infection. At present it is probably justifiable to use these antibiotics only in severe cases, such as ophthalmic zoster, or where a potentially fatal complication such as meningo-encephalitis has occurred.

SUMMARY

A case of herpes zoster presenting with auricular eruption, facial paralysis and encephalitis is reported. The pleomorphism of the disease is noted. Herpetic encephalitis and the Ramsay Hunt syndrome are discussed. The use of the term " geniculate herpes " is deprecated. It is suggested that treatment with antibiotics might be reserved for severe cases and for those with serious complications.

I wish to thank Dr. A. M. G. Campbell for his opinion, Dr. F. J. W. Lewis for the pathological report and Dr. J. A. Birrell for permission to publish.

REFERENCES

Biggart, J. H., and Fisher, J. A. (1938). *Lancet*, **2**, 944.

Binder, M. L., and Stubbs, L. E. (1949). *J. Amer. med. Ass.*, **141**, 1050.

Brain, W. R. (1951). *Diseases of the Nervous System*, 4th ed., 491. London.

Carter, A. B. (1951). *Brit. med. J.*, **i**, 987.

Critchley, M. (1947), in *The Medical Annual*, 281. Bristol.

Denny-Brown, D., Adams, R. D., and Fitzgerald, P. J., (1944). *Arch. Neurol. and Psychiat.*, **51**, 216.

Finland, M., Finnarty, E. F., Jr., Collins, H. S., Baird, J. W., Gocke, T. M., and Kass, E. H., (1949). *New. Engl. J. Med.*, **241**, 1037.

Foster, J. H., and Jackson, A. H. (1951). *Conn. med. J.*, **15**, 199, quoted in *Excerpta Medica* (Internal Medicine), (1951), **5**, 1482. Amsterdam.

Hunt, J. Ramsay (1907). *J. nerv. ment. Dis.*, **34**, 73.

Kass, E. H., Aycock, R. R., and Finland, M. (1952). *New Engl. J. Med.*, **246**, 167.

Parkinson, T. (1948). *Brit. med. J.*, **1**, 8.

Schiff, C. I., and Brain, W. R. (1930). *Lancet*, **2**, 70.

HERPES ZOSTER GENERALISATUS

BY

MARK HEWITT, M.B., M.R.C.P.

Senior Registrar, Department of Dermatology, United Bristol Hospitals

Herpes zoster is usually a mild illness with a characteristic painful rash and slight constitutional disturbance. The eruption is confined in the majority of cases to one or two posterior nerve root segments but a few aberrant vesicles are commonly found in the vicinity. Occasionally several nerve roots are affected and one case of universal segmental involvement has been described (Colombini, 1893).

Rarely, a generalized vesicular eruption identical with varicella complicates herpes zoster. It has been called herpes zoster generalisatus (H.Z.G.) or herpes zoster varicellosus. Four cases in chronically ill patients are described in this paper. Clinically, chicken-pox and herpes zoster are closely related and are probably caused by the same virus. Numerous cases of cross-infection have been reported; the usual sequence is that chicken-pox follows contact with zoster, the reverse being rarer. Herpes zoster generalisatus has been considered a dual infection with zoster and varicella.

Experimental support for the concept that a single virus is responsible has been provided by Brain (1933) by cross complement fixation reactions, and by Blank *et al.* (1951) who showed that in scrapings from the floor of vesicles, fixed and stained with Giemsa, the nucleoli of the giant multinuclear epithelial cells are replaced by amorphous matter and melanin granules; unique changes in zoster and varicella which cannot be demonstrated in other virus infections of skin. Brain suggests that the virus has neurotropic and dermatotropic properties which may be modified by unknown factors.

Physical weakness with a lowered resistance to infection may act as a modifying factor—it is a prominent feature in each of these cases in whom a severe attack of herpes zoster was complicated by herpes zoster generalisatus.

CASE 1

Rheumatoid Arthritis. Chronic Bronchitis. Prostatic Obstruction. H.Z.G.

A bus-driver, aged fifty-nine, was referred by Dr. P. S. Sinclair on account of severe stabbing pain in the right upper quadrant of his back followed three days later by a profuse haemorrhagic vesicular eruption in the second right thoracic nerve segment. Many of the vesicles became confluent and necrotic [*Plates XI and XII*], and finally cleared with extensive scarring. Between the third and fourth day after the onset of the segmental eruption several crops of isolated vesicles appeared on the face, head and neck, trunk, legs and arms. His general condition was poor, he was prematurely aged, his lips were cyanosed and he was dyspnoeic on exertion. He had a severe dorsal kyphosis; the thoracic cage was barrel-shaped and rigid, the chest expansion was limited to half an inch, and sibilant rhonchi were audible in all areas. There had been intermittent symptoms of prostatic obstruction for two years and he now developed acute retention of urine, the blood urea

rising to 68 mgm. per cent. The urine was not infected and cystoscopic examination failed to reveal herpetiform lesions in the bladder, although these have been reported (Dubois, 1926) in a case of herpes zoster of the left buttock. For nine years he had been receiving treatment for chronic rheumatoid arthritis. At the time of examination the arthritis was not active but there were severe periarticular swelling, limitation of movement and chronic deformity of the hand and of the wrist, elbow, shoulder, and knee joints. Large subcutaneous nodules were present on the extensor aspects of both forearms. Biopsy revealed areas of necrobiosis and round-cell infiltration typical of those found in chronic rheumatoid arthritis.

CASE 2

Parapsoriasis. Acute Gastric Ulcer. H.Z.G.

A labourer, aged sixty-eight, a patient of Dr. B. E. McConnell, had noticed symptomless scattered brown pigmented scaly erythematous patches on his chest six years earlier, which had gradually increased in size and number and had become widely distributed on his trunk, legs and arms. A diagnosis of parapsoriasis was made. For three years he had each winter complained of epigastric pain made worse by food and radiating upwards to the chest and down to the abdomen and through to the inter-scapular area. The pain, with flatulence, usually lasted for eight weeks, clearing only after rest, diet and alkalis.

While undergoing treatment for a recurrence of this dyspepsia, he developed herpes zoster and was referred to the dermatological unit by Dr. G. E. F. Sutton on 10th December, 1950.

The primary lesions involving the right first and second lumbar segments had started two days previously on the thigh, pubic area and buttock. There was a profuse eruption of vesicles arranged in clusters, many were confluent and presented a linear distribution. Slightly less extensive vesiculation involving a similar area had developed in the left first and second lumbar segments. Large numbers of isolated scattered vesicles were present over the remainder of the trunk, the limbs, and the head and neck—those nearest to the primary lesions being most profuse. Fourteen days after its onset the rash had cleared.

His general condition was unsatisfactory; he had been ill at home with dyspepsia seven weeks prior to admission and had lost one stone in weight. The haemoglobin was 13·3 grams per cent. A barium meal revealed a large ulcer crater high up on the lesser curvature of the stomach. On 30th December there was severe melaena with shock and collapse, and on 4th January, 1951, the red-cell count had fallen to 1,600,000 per c.mm. and the haemoglobin to 3·7 grams per cent. Thereafter he made an uninterrupted recovery.

A fractional test meal revealed a histamine fast achlorhydria but a further barium meal and gastroscopic examination excluded a neoplasm of the stomach. The H.Z.G. had supervened in a severely ill patient with a chronic gastric ulcer. The parapsoriasis may or may not have influenced the extension of the herpes zoster.

CASE 3

Hodgkin's Disease. Gastric Ulcers. H.Z.G.

A housewife, aged thirty-three, a patient of Dr. B. L. Hodge, noticed a lump above her left clavicle in August, 1947, and a biopsy revealed Hodgkin's disease. Until her death in February, 1951, she was admitted to hospital on eight occasions for deep X-ray therapy to metastatic deposits in the neck, axillae, chest, supra and infra clavicular areas, sacral region, groins, the dorsal spine and sternal area. In April, 1950, she complained of pain in her back, and sclerotic metastases were detected radiologically in the 3rd, 5th, 6th, 7th and 8th thoracic vertebrae. These bones were irradiated in May, 1950, by which time she was emaciated and anaemic, and her general condition was deteriorating.

On 18th October, 1950, shortly after readmission she developed herpes zoster of the eighth left thoracic segment with severe pain and pruritus, a general constitutional disturbance and a raised temperature. On 23rd October a fresh crop of vesicles appeared in the left axilla and on the following day large numbers of vesicles developed on other areas of the chest, trunk, arms and legs. All the lesions had faded by 10th November, 1950.

A month later ascites began to complicate existing hepatic and splenic enlargement, and there were irregular palpable masses in the epigastrium and right hypochondrium.

PLATE XI

Herpes Zoster Generalisatus.

PLATE XII

Herpes Zoster Generalisatus.

She began to pass large quantities of blood per rectum, and died on 24th February, 1951.

A post-mortem examination revealed gross fibrous enlargement of the para-aortic glands and large masses of fibrous tissue in the portal fissure compressing the portal vein. The liver weighed 2,270 grams, had a pitted surface, was unusually lobulated, and contained irregular areas of growth. In the stomach there were two chronic ulcers one inch and half an inch in diameter on the middle third of the lesser curvature, two small chronic ulcers in the prepyloric region and one three-quarters of an inch in diameter on the posterior wall. In the chest the tracheobronchial glands appeared normal but were invested with large masses of fibrous tissue which in one area were constricting the left upper bronchus and producing collapse of the lung. The rest of the lungs were oedematous.

CASE 4

Seminoma Testis. H.Z.G. Herpes simplex.

A labourer, aged thirty-five, noticed a painful swelling of the left testicle in June, 1950. Orchidectomy was performed on 1st August, 1950, and the diagnosis of seminoma was confirmed histologically. Three weeks later there was radiological evidence of lung metastases, and the chest and abdomen were treated with deep X-rays. The deposits decreased, but in November, 1950, he was readmitted on account of severe unproductive cough. Skiagrams revealed an increase in size of the lung metastases. His symptoms were immediately relieved by deep X-ray therapy and he was discharged home for a few days at Christmas.

On readmission for completion of treatment on the 2nd January, 1951, he was found to have herpes zoster. On that day, a painful pruritic eruption had developed on the left side of the chest in the second and third thoracic segments; there were clusters of vesicles of herpes simplex on the lower lip. On 7th January the eruption became generalized and vesicles appeared on the legs, trunk, face and neck and soft palate. On 9th January his general condition was worse, the eruption more profuse and, although older vesicles were fading, numerous fresh crops were appearing. The patient was dyspnoeic with a temperature of 102° F. which steadily rose to 105° F. on 11th January when he became comatose and died. H.Z.G. and bronchopneumonia were terminal infections.

Post-mortem examination showed that death was due to bronchopneumonia and myocardial failure. Herpes zoster scars were present over the left side of the chest, and more recent herpetic lesions were scattered on the face, back and legs. There was no superficial glandular enlargement. The liver contained a few firm nodules of secondary growth. The histological changes in the skin lesions were simply those of herpes zoster. The metastases consisted of a solid alveolar structure composed of large round or oval cells with a single large nucleus and a pale cytoplasm. There were numerous mitoses.

DISCUSSION

Aberrant vesicles commonly accompany the zonal eruption of herpes zoster and are identical with the isolated vesicles of herpes zoster generalisatus. Tenneson (1893) described aberrant vesicles in nine out of ten cases of herpes zoster. Bluefarb and Morris (1941) found them in twenty out of thirty cases, and believed that they were commoner in elderly patients, particularly those with a severe infection.

Since Lippe (1889) first described a generalized vesicular eruption with herpes zoster numerous cases have been reported. Many patients have been elderly, others have had serious physical disabilities [Parounagian and Goodman (1923), Grindon (1939)]. Nobl (1911) and later Skeer (1936), Wile and Holman (1940) believe that herpes zoster generalisatus is a specific complication of chronic leukaemia, and indeed many patients with chronic lymphatic leukaemia who later developed herpes zoster generalisatus had previously received deep X-ray therapy. Ellis and Stoll (1949) consider that the effects of this treatment predispose to herpes zoster in irradiated posterior nerve roots.

Two of the present cases had been irradiated over spinal root areas which shortly afterwards became the site of severe herpes zoster. Grave physical disabilities are a common factor in the cases described, indeed in one bronchopneumonia and herpes zoster generalisatus were terminal. The effects of age in two and debility in all four may have played a major role in the general spread of the zonal lesions. None of these patients had been exposed to herpes zoster or varicella and no contacts were infected.

If, as seems likely, herpes zoster is due to a neurotropic factor and the generalized eruption is due to a dermotropic factor of the same virus, the clinical features of these cases suggest that age, debilitating illness, and X-rays may in some way modify the activity of the virus.

SUMMARY

Four patients with herpes zoster generalisatus are described. Each had a severe initial infection with zoster, and other serious physical disabilities may have been responsible for spread of the eruption.

I am indebted to Drs. S. Curwen and R. P. Warin for permission to report these cases; to Dr. A. L. Taylor for details of the post-mortem findings, and to the Photographic Department of the United Bristol Hospitals.

REFERENCES

Blank, H., Burgoon, C. F., Baldridge, G. D., McCarthy, P. L., Urbach, F. (1951). *J. Amer. med. Assn.*, **146**, 1410.

Bluefarb, S. M., and Morris, G. E. (1941). *Arch. Derm. Syph.*, **43**, 385.

Brain, R. T. (1933). *Brit. J. exp. Path.*, **14**, 67.

Colombini (1893). " Caso singolarissimo di Herpes zoster universale." Siena. Quoted by Blaschko, A. (1902), " Herpes ", in Mracek, F. (ed.), *Handbuch der Hautkrankheiten*, Vol. I, pp. 677–722. Wien, 1902.

Dubois, F. E. (1926). *J. Urol.*, **15**, 583.

Ellis, F., and Stoll, B. A. (1949). *Brit med. J.*, **2**, 1323.

Grindon, J. (1939). *Arch. Derm. Syph.*, **39**, 865.

Lippe (1889). Quoted by Schonfeld, W. (1928), " Zoster und Herpes Simplex ", in Jadassohn, J. (ed.), *Handbuch der Haut- und Geshlechtskrankheiten*, Bd. 7, Teil 1, p. 1. Berlin.

Nobl, G. (1911). " Zur Kermtnis des Herpes zoster generalisatus ", *Wien Klin. Wschr.*, **24**, 14.

Parounagian, M. B., and Goodman, H. (1923). *Arch. Derm. Syph.*, **7**, 439.

Skeer, J. (1936). *Arch. Derm. Syph.*, **34**, 809.

Tenneson, H. (1893). *Traite clinique de dermatologie*, 116. Paris.

Wile, U. J., and Holman, H. H. (1940). *Arch. Derm. Syph.*, **42**, 587.

Correspondence

SIR,

We are always hearing about the long time patients have to wait in the Out-Patient Department in Hospitals. Do many of those who sit for long hours on the hard benches really need to be there at all? Was their journey really necessary? I refer to the large number of follow-up cases who will in the end only be seen by a Junior Registrar or perhaps a newly qualified House Surgeon, who will examine their operation scar and satisfy himself that the wound has healed by first intention. Surely the General Practitioner who has first referred the case to the Physician or Surgeon is quite capable of carrying out the follow-up care himself, and referring the patient again to Hospital if he is not satisfied with the progress.

Some of us are becoming tired of the " pinching " of the interesting work which falls to our lot. Is it too late to convince our Consulting Surgeons and Physicians that most General Practitioners are quite capable of doing much of the work which at present clutters up the Out-Patient Departments of our Hospitals? When the G.P. sends a patient up for an opinion he means an *OPINION* as to diagnosis and treatment, and not that the patient shall be taken over permanently by the hospital.

D. H. FORSTER

COLLEGE OF GENERAL PRACTITIONERS

South Western Regional Faculty

In January, 1953, the College was launched, and it quickly became apparent that it was being supported. The Foundation Council from the beginning realized that it must differ from other colleges in that its work must be done in regions, preferably centred on universities. When over a hundred from the South West had become foundation members, it was decided to call a meeting in Bristol. On 28th June, 1953, sixty-four members from the counties of Bristol, Cornwall, Devon, Dorset, Gloucester, Somerset and Wilts met in the Board Room of the Bristol Royal Infirmary. Dr. John Hunt, honorary secretary of the College, addressed the meeting, and after full discussion, it was resolved to form a South Western Regional Faculty. A Board was thereupon elected, consisting of three members from each of the seven counties, and this Board met after the general meeting, and elected as its Chairman Dr. J. H. Grove-White of Cirencester, and as its Vice-Chairman Dr. R. M. S. McConaghey of Dartmouth. It held its second meeting in October, and will meet again in December. Dr. A. H. Gale, Director of Medical Postgraduate Studies at the University of Bristol, was present by invitation at the inaugural meeting of the Faculty, and has been most helpful. Mr. John Dodd has very kindly taken on the duties of Honorary Assistant Secretary.

The aims of the College are not political. They are to watch over the academic interests and education of general practitioners. The Report of the Steering Committee says it " can find no evidence that any existing administrative body

in this country is doing now, or will be able to do in the future, what practitioners require ". The main committees of the Foundation Council are concerned with Undergraduate Education, Postgraduate Study and Research. These three subjects will also be the main concern of the Regional Faculties. Any general practitioner who wishes to join the College should write to me.

D. M. CAMERON,
Hon. Sec.

News from Societies

BRISTOL MEDICO-CHIRURGICAL SOCIETY

The Annual Clinical Meeting of the Society was held at the Bristol Royal Infirmary on 11th November, 1953.

The meeting was well attended and there were nearly forty cases and demonstrations. It is not within the scope of this article to summarize every case, but several interesting and important points were emphasized in the discussion at the end of the meeting.

Professor Neale discussed an excellent demonstration of chest deformities in childhood. Now that rickets has virtually been eliminated, it is even more apparent that there are a number of other causes of malformed chests, such as congenital heart disease and congenital anomalies of the thoracic spine and ribs. Professor Neale emphasized the importance of establishing the correct aetiology in these cases.

Mr. Pridie demonstrated eight cases of arthrodesis of the hip by central dislocation and made the following points most clearly:

Cases of Osteoarthritis of the hip should not be treated surgically until the patient is unable to work through pain and disability. Arthrodesis of the type demonstrated is the operation of choice. After such a procedure, not only is pain abolished but commonly the patient returns to his occupation which may be most arduous. Arthroplasty with the use of acrylic cups is not a satisfactory procedure and is rapidly falling into disrepute.

Dr. Apley commented on some cases of pseudo-hermaphroditism and pointed out that these conditions are much commoner than the majority of us would suppose. He emphasized the importance of early diagnosis of the true sex so that these unfortunate individuals may be brought up in their correct sex. The true sex can always be determined and practitioners should never hesitate to seek specialist advice in these difficult cases, and while treatment is often unsatisfactory many social tragedies can be avoided by correct diagnosis.

Dr. Cosh spoke on some aspects of diabetic complications with special reference to neuropathy and retinopathy. The latter condition was being seen much more frequently nowadays because many more diabetics live to a ripe old age and **Mr. Garden** pointed out that not only is diabetic retinopathy common, but its course is unaffected by insulin therapy.

The new insulin zinc suspensions were also discussed and will doubtless become a useful addition to the range of insulins. However, in the light of present knowledge they should not be used for haphazard replacement of well tried preparations now in everyday use.

Mr. Horton commented on some interesting cases of peripheral vascular disorders treated surgically and it is very gratifying to learn that major reconstruction operations in this field of surgery are proving highly satisfactory.

A most instructive evening ended with acclamation of all those who had gone to considerable trouble to make the meeting such a success.

SOME REFLECTIONS

BY RIP VAN WINKLE, M.D.

" You know what that is? In a certain place
meet certain doctors to discuss a case
And other matters, such as weather, crops,
potatoes, pumpkins, lager-beer and hops."

OLIVER WENDELL HOLMES

And that, of course, is the trouble with clinical meetings. I don't say I actually spent much time discussing pumpkins or lager-beer, but by the time I'd had a chat with B., whom I haven't seen for a long time, and asked J. about that case I'd sent him, there didn't seem much time left for looking at the patients.

I heard someone say afterwards it is " rather like an agricultural show; you can only hope to see a few of the exhibits ". Certainly all the stalls and sideshows seemed to be doing good business. The insulin exhibits attracted great interest and were, I thought, very clear and concise. The Children's Hospital's collection of chest deformities, was the perfect subject for this type of meeting, and was very ably presented.

The refreshments, which gave us a little more time to discuss the equivalents of Oliver Wendell Holmes's weather, potatoes and hops, were most commendably up to standard. The discussions after were . . . well, shall we say, not quite up to the refreshments, but they gave one an opportunity to compare the oratorical styles of one's colleagues.

There was Professor Neale, never still, very erudite, and, to one listener at least, a little incomprehensible; Mr. Pridie, forthright, an enthusiast for his own viewpoint and therefore putting it over well, and finally propounding the great truth, not always remembered, that the only good result is an active working man or woman. Dr. Apley, in conversational style and with the air of one about to be delivered of a slightly *risqué* story: Dr. Cosh, elegant, quiet, economical in words and gestures, and supported by Dr. Ferguson and others in, almost, warning us off the new insulins, these interesting substances being still, as it were, *sub judice*: lastly, Mr. Horton, in physicianly rather than surgeon's style, quietly apologizing that his best case, misdiagnosed and given up as hopeless in the U.S.A., had been discharged cured and could not be shown: and so to the President charmingly enthusing over a " beautiful pair of discs ", before closing the meeting with a word of thanks to the organizers which this humble scribe sincerely applauded.

What do the patients at these shows think of it? I've often wondered: now I have one answer at least. As Dr. X left one cubicle, I overheard him say " Extraordinary case, doesn't look a bit like a . . . " I went in and heard the patient whispering to someone with him, " Funny-looking bloke, doesn't look like a doctor at all".

" MALIGNANT DISEASE: THE PROBLEM OF THE ADVANCED CASE "

Mr. R. V. Cooke, M.Ch., F.R.C.S., spoke to the Bristol Medico-Chirurgical Society at the December meeting on *Malignant Disease: The Problem of the Advanced Case.*

The gradual rise in age of the population is giving larger numbers of sufferers from certain cancers—particularly large bowel—while the breast is still a major problem in women in the prime of life. Here, however, the recent endocrine treatments are an advance—testosterone for the pre-menopausal woman and oestrogens for the elderly. Mr. Cooke also referred to adrenalectomy, which appears to hold hope for the very advanced case with metastases. Oestrogens are of course well known in the treatment of prostatic carcinoma.

The thyroid is a less common problem and would be rarer still were surgery used earlier in goitre swellings. Radioactive iodine held great hopes at first, but it cures only

about one case in ten. With endocrine therapy in general, as **Dr. Foss** reminded us later, expense can be formidable and each case needs very careful thought.

Mr. Cooke paid tribute to radiotherapy, to be used by means of close co-operation between all workers.

On the subject of bowel cancer the speaker turned to the purely surgical aspect. Here only good and radical excision can offer hope. He showed us inspiring examples of what could be achieved even before the recent advances in anaesthesia and antibiotic therapy.

Even to-day, surgery it seems still has need of the lion hearted.

Appointments

SOUTH WESTERN REGIONAL HOSPITAL BOARD

CONSULTANTS, SPECIALISTS AND REGISTRARS APPOINTED DURING THE PERIOD OCTOBER TO DECEMBER, 1953

BRISTOL

Name	*Appointment*	*Formerly*
CHAMPION, ELIZABETH P. (Mrs. Bassett), M.B., B.S., (SYDNEY), D.A. (LONDON).	Anaesthetic Registrar, Southmead Hospital.	Locum Consultant Anaesthetist, Harts Hospital, Woodford.
ILES, KATHLEEN C., M.B., CH.B.(BRISTOL).	Registrar in Diseases of the Chest, Bristol.	J.H.M.O., Royal National Hospital for Diseases of the Chest, Ventnor, I.O.W.
RICHARD, D. R., B.M., B.CH.	Surgical Registrar, Weston-super-Mare General Hospital.	Locum S.H.O. in General Surgery, St. Alfeges Hospital, Greenwich.
ARTHURS, G., L.R.C.P., M.R.C.S., M.B., B.S., F.R.C.S.	Senior Registrar in Neurosurgery, Frenchay Hospital.	Senior Registrar in Neurosurgery, Morriston Hospital, Swansea.
DUTTON, J. E. M., M.B., B.S., F.R.C.S.	Registrar in Neurosurgery, Frenchay Hospital.	S.H.O., Neurosurgical Unit, Frenchay Hospital.
SEAGER, C. P., B.SC., M.B., B.CH.	Psychiatric Registrar, Bristol Mental Hospitals.	S.H.O., Bristol Mental Hospitals.
JOYCE, M. H. B., M.B., CH.B.	Psychiatric Registrar, Bristol Mental Hospitals.	S.H.O., Bristol Mental Hospitals.
HEATON-WARD, W. A., M.B., CH.B. (BRISTOL), D.P.M. (BRISTOL).	Medical Superintendent, Stoke Park Colony Group.	Deputy Medical Superintendent, Hortham–Brentry Hospital Group.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
MARTIN, L. V., M.B., B.S., D.A.	Consultant Anaesthetist, North Glos. Clinical Area.	Senior Registrar in Anaesthetics, Welsh National School of Medicine, Cardiff.
FOREMAN, J. M., M.B., CH.B. (NEW ZEALAND), F.R.C.S.E.	Surgical Registrar, Glos. Royal Hospital.	Surgical Registrar, Newcastle General Hospital.
SANDERS, E. J., M.B., B.CH. (CAPE TOWN).	Surgical Registrar, Cheltenham General Hospital.	H.S., General Surgical Unit, Oldcurch Hospital, Romford.
VINCENT, C. T., M.R.C.S., L.R.C.P., M.B., B.S.	Assistant Clinical Pathologist (S.H.M.O.), North Glos. Clinical Area.	Senior Pathological Registrar, Lambeth Group Laboratory.

BATH

Name	*Appointment*	*Formerly*
WELLS, C. E. C., M.B., B.S., M.R.C.P.	Senior Medical Registrar, Bath Clinical Area.	Medical Registrar, Southmead Hospital.
MUSCAT, S., B.SC. (MALTA), M.D. (MALTA), D. (OBST.), R.C.O.G.	Registrar in Obstetrics and Gynaecology Bath Group of Hospitals.	S.H.O., Obstetrics and Gynaecology Dept., Farnborough Hospital.
MORGAN, JEAN E., M.B., CH.B.	Pathological Registrar, Bath Group of Hospitals.	S.H.O., Dept. of Pathology, Frenchay Hospital.

SOUTH SOMERSET

Name	*Appointment*	*Formerly*
PRETTEJOHN, E. J. T., M.B., B.CHIR., M.R.C.P.	Consultant Dermatologist, South Somerset Clinical Area (three sessions).	Senior Registrar in Dermatology, United Bristol Hospitals.
HARKNESS, J., B.SC., M.B., CH.B., M.D.	Area Pathologist, South Somerset Clinical Area.	Consultant Pathologist, Central Laboratory, Portsmouth.
CUTHILL, J. M., M.B., CH.B., D.P.M.	Senior Registrar in Psychiatry, Tone Vale Hospital.	Deputy Specialist i/c Neuropsychiatric Centre, Princess Mary's R.A.F. Hospital.

EXETER

Name	*Appointment*	*Formerly*
FREEMAN, A. G., M.R.C.S., L.R.C.P., M.A., M.B., B.CHIR., M.D., M.R.C.P.	Senior Medical Registrar, Exeter Clinical Area.	Assistant Chest Physician (Locum), Cardiff and District.
CANDLER, T. O., M.B., B.CH., M.R.C.S., L.R.C.P., F.R.C.S.	Clinical Assistant in Orthopaedic and Traumatic Surgery, Bideford and District Hospital (one session).	In general practice, Bideford.

WEST CORNWALL

Name	*Appointment*	*Formerly*
PENGELLY, C. D. R., M.B., CH.B., M.R.C.P.E.	Senior Medical Registrar, West Cornwall Clinical Area.	Medical Registrar, United Bristol Hospitals.
LAKEMAN, MARGARET A., M.R.C.S., L.R.C.P., D. (OBST.), R.C.O.G., M.B., B.S.	Registrar in Obstetrics and Gynaecology, Camborne–Redruth Miners' and General Hospital, Redruth.	H.S., Whittington Hospital, London.

Notes and News

POSTGRADUATE COURSES FOR GENERAL PRACTITIONERS

The following courses are being arranged:

1. *May 14th to 17th:* Week-end course at Exeter.
2. *July 11th to 17th:* Intensive course at Bristol in Medicine, Surgery and Paediatrics.
3. *April–June:* Sunday morning lecture demonstrations in Bristol.

Detailed programmes may be obtained later from: Director of Postgraduate Medical Studies. Telephone: Bristol 24161. Extension 7.

MEDICAL EXHIBITIONS

The Organizers of the London Medical Exhibition have recently announced plans for their 1954 provincial Exhibition, to be held at Victoria Rooms, Bristol University, from May 17th to 21st, inclusive.

Information relating to this and the London Exhibition should be obtained direct from: The Organizers, London Medical Exhibition, 194–200, Bishopsgate, London, E.C.2.

WESSEX RAHERE CLUB

The Spring Dinner of the above Club will take place at the Royal Clarence Hotel, Exeter, on Saturday, April 24th.

Mr. H. Jackson Burrows, F.R.C.S. has kindly accepted the invitation to be present as Guest of Honour.

Membership of the Club is open to all Barts men practising in the West Country. Further details will be circulated to Members and to any other Barts men who are interested and who will get in touch with the Hon. Secretary, Mr. A. Daunt Bateman, of 11 The Circus, Bath.

BAILBROOK HOUSE · BATH

Tel. Batheaston 88189

FOR the treatment of Voluntary, Temporary and certified patients suffering from Nervous and Mental Disorders.

10 minutes from Western & Midland Region Railway Stations. 2 hours from Paddington.

Terms from 11 guineas a week

Resident Physician

A. GUIRDHAM, M.A., D.M., B.Sc. (Oxon), D.P.M. (London)

MOUNT PLEASANT

and

ST. MARGARET'S

WALTON, CLEVEDON

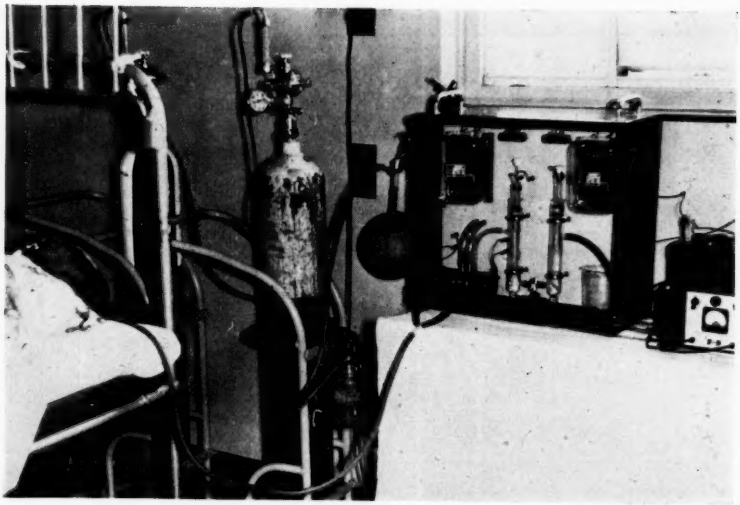
NURSING HOMES for the MEDICAL TREATMENT of mild nervous and other illnesses, by means of psycho-therapy, E.C.T., occupational therapy, exercises, &c.
Special geriatric care provided.

Terms from £6 6s. a week inclusive
Patients can attend daily for treatment

Visiting Psychiatrist: GERALD DE M. RUDOLF, M.R.C.P., D.P.M., D.P.H.

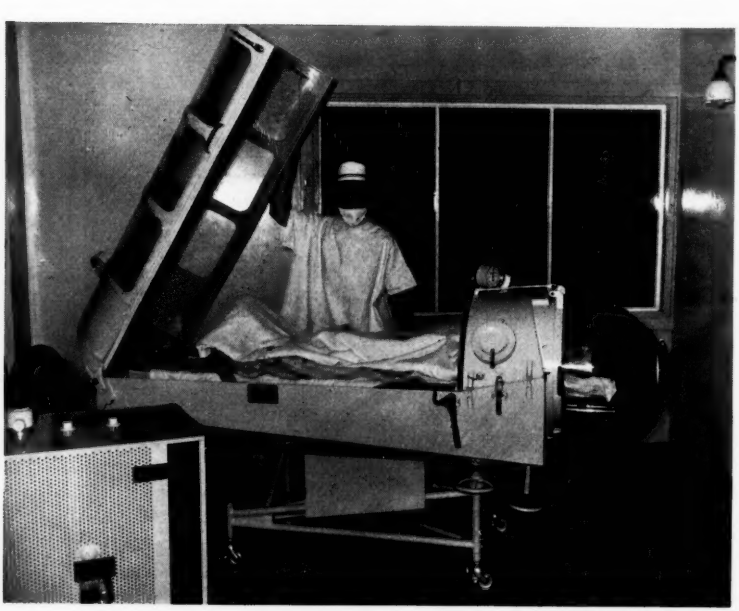
ELIZABETH CASSON, O.B.E., M.D., D.P.M.
St. Margaret's, Walton, Clevedon, Som.
Telephone: Clevedon 627

PLATE XIII



Clevedon Positive-Pressure Respirator

PLATE XIV



Bristol Tank Respirator

Editorial

RESPIRATOR RESEARCH AT HAM GREEN HOSPITAL

Breathing difficulties in poliomyelitis are most commonly due either to weakness of the respiratory muscles or to obstruction of the upper respiratory passages by secretions. The first group will usually respond to treatment in a tank-type of respirator and the second to postural drainage combined with suction. There remains a group of cases with mixed spinal and bulbar involvement which had a very high mortality until two years ago when Lassen[1] introduced a new method of treatment during the severe epidemic of poliomyelitis in Copenhagen. This involves a tracheotomy with insertion of a cuffed tube which blocks off the pharynx and its secretions and through which the lungs are regularly inflated by positive pressure. Lassen used relays of medical students to supply the pressure.

Working at Ham Green, Macrae and McKendrick have developed an automatic machine, the *Clevedon Positive-Pressure Respirator* (Plate XIII), to take the place of the medical students and have reported in the *Lancet* their experience with their first case.[2] The patient, a man of twenty-one, would undoubtedly have died without this new method. Progress has so far been very satisfactory, power of swallowing returning after six days and some spontaneous respiration beginning soon after. Forty-seven days after the start of positive-pressure respiration the tracheotomy tube was finally removed and the wound allowed to heal. The patient has now been home for more than a month and is able to walk and look after himself.

It is believed that this is the first case of poliomyelitis to be treated by this method in this country. Although the difficulties of management have not been so great as was feared close supervision is vital and at Ham Green it has been considered essential to have a doctor on the ward twenty-four hours a day. The method is therefore one which can only be used in well-equipped hospitals with adequate medical staff.

Many difficulties have had to be overcome to produce a satisfactory respirator within a few months and the workers at Ham Green are to be congratulated.

Last year it was also apparent that the manufacture and design of tank respirators in this country had not moved appreciably since Lord Nuffield presented the *Both* respirator to the Nation in 1938. It seemed, therefore, appropriate to try to utilize the wealth of mechanical skill available in the Bristol area to design and build a prototype tank respirator on entirely new lines.

The subject was studied at Ham Green, and the co-operation of the Bristol Aeroplane Company was sought and readily obtained. In August, 1953, the South Western Regional Hospital Board authorized the necessary expenditure, and the apparatus was completed in December.

The *Bristol Tank Respirator* (Plate XIV) is now installed at Ham Green Hospital and has already been used in the care of several patients.

The primary object of the design is ease of nursing combined with comfort for the patient. Construction is largely in light alloy and perspex, and a pleasing appearance has been obtained quite unlike any previous type of tank respirator. The machine provides for positive-pressure breathing through the mouth as well as conventional negative-pressure cabin respiration, and longitudinal and lateral tilt can be simply and accurately adjusted. The speed of breathing and volume of tidal air can be varied within wide limits.

The development of this machine owes much to the interest and enthusiasm of members of the staff of the Bristol Aeroplane Company. It has been very successful in use and one has been ordered for use in the Bristol United Hospital.

[1] Lassen, H. C. A. (1953). *Lancet*, i, 37.
[2] Macrae *et al.* (1953). *Lancet*, ii, 971.
[3] *Lancet*, 1954, April 3rd.

CHANGES IN THE EDITORIAL COMMITTEE

Until he was elected President of the Society, Dr. Sutton had served us as treasurer both of the *Journal* and of the Society since 1946. He was also the senior member of the editorial committee and did much to keep the *Journal* alive in the last few years.

During the year we have also lost the services of Dr. Middlemiss, Dr. MacLachlan and Dr. Wood. The first two are now unable to spare the time. Dr. Middlemiss, in fact, served two months abroad as Adviser on radiology for the Colonial Office and his account of his journey is included in this issue. Dr. MacLachlan joined us three years ago and proved a stimulating and imaginative colleague. He now finds that the burdens of a city councillor and of a busy practitioner do not leave him time to do his editorial work to his satisfaction.

Dr. F. J. Y. Wood has gone to Southampton and we will miss him as editorial secretary very much.

DAMNED DRUGS

PART I

The Presidential Address, given at The Royal Fort, Bristol, on 8th October, 1952 at the opening of the Seventy-Fourth Session of the Bristol Medico-Chirurgical Society

BY

DR. G. E. F. SUTTON, *M.C.*, M.D., F.R.C.P.

Consultant Physician, Bristol Royal Hospital

In *Cymbeline* the king's physician thus describes the love of the former queen:

> More sir, and worse, she did confess she had
> For you a mortal mineral which being took
> Should by the minute feed on life and lingering
> By inches waste you; In which time she proposed
> By watching, weeping, tendance, kissing, to
> O'ercome you with her show.

Even so throughout the long and chequered history of man's career on " this darkling plain swept with confused alarms of struggle and flight", there have been men and women who have brewed strange brews, either to deal death swift and sudden, or to encompass their victim more slowly and even more insidiously than the silent march of some fell disease.

Poisons were well known among the ancients, and centuries before the Christian Era the Sanskrit writings show that the Hindoos were familiar with animal, vegetable and mineral poisons as well as with their antidotes. Thus it was written: " It is necessary for the practitioner to have knowledge of the different poisons and antidotes, as the enemies of the Rajah—bad women and ungrateful servants—sometimes mix poison with food. Food which is thus suspected should first be given to certain animals and if they die it is to be avoided."

The most notorious as well as the most remorseless of the Greek poisoners was Olympias, the wife of Philip of Macedon and mother of Alexander the Great. Among many others, she was responsible for the death of Aridaeus, the natural son of Philip, his wife Eurydice and Nicanor. To those who were imprisoned at her instigation she kindly allowed the choice between the sword, a rope, and a cup of hemlock.

Later, in the first century of the Christian Era, we find the loathsome Locusta using slow and wasting poisons to destroy her numerous victims. She dispatched Claudius by order of Agrippina, and, later, Britannicus by order of Nero. Indeed the latter was so impressed by her talent that he showered gifts upon her and insisted on her teaching a selected class in order that her art might be perpetuated.

In the year 331 B.C., an epidemic broke out in Rome which was at first supposed to arise from corrupt air until it was observed that the illness was confined to the principal patricians. Even so, further inquiries would not have been made were it not for the sudden and unexpected accusation by a female slave of more than twenty Roman ladies of poisoning. There followed a widespread investigation and inquiry, as a result of which no less than 366 ladies were accused of poisoning. Two of them, Cornelia

and Sergea, both distinguished patricians, were caught in the act of preparing their fatal potions, but, when they were led before the popular assembly, they swore that their preparations were but harmless remedies and promptly accused the slave of being a false witness. She, however, had the wit to suggest that in proof of their statement the ladies should swallow these so-called harmless remedies themselves. This they did and were thus fortunate in avoiding a painful trial and a more shameful death.

Turning over the pages we next dip into the fifteenth century in Italy where flourished the notorious Venetian poisoners. The infamous Council of Ten here met to consider the removal of emperors, princes, powerful nobles and other high dignitaries by the subtle and time-honoured method. Records still exist of their proceedings and they show that whenever the deed was done the simple and significant word " Factum " was appended to the victim's name.

On December 15th, 1643, John of Regula, a Franciscan friar, offered the Council of Ten a selection of poisons and calmly informed them that he would remove anyone they desired for a suitable sum. His terms were clearly and succinctly stated as follows: " For the first successful case a pension of 1,500 ducats a year, to be increased on the execution of the projected task." His tariff then ran as follows: " For the Great Sultan 500 ducats. For the King of Spain 150 ducats (including expenses of the journey). For the Duke of Milan 60 ducats. For the Pope 100 ducats." An addendum to his tender ran as follows: " The farther the journey and the more eminent the man, the more it is necessary to reward the toil and hardship undertaken, and, accordingly, the heavier must be the payment."

The Presidents of the Council, Guolando Duoda and Pietro Guiarini, placed his project before the members on January 4th, 1544, and on a division it was resolved to accept this patriotic offer and to experiment first on the Emperor Maximilian.

From the sixteenth century onwards, poisoning in Italy assumed endemic, and, at times, even epidemic proportions. The most notorious exponent at this time was the woman Toffana, or Toffania, who made a fortune from dispensing poisons throughout Italy. Small glass vials were dispatched bearing the image of a saint and labelled with various attractive names such as " Aquetta di Napoli " or " The Manna of St. Nicholas of Bare " and " Aqua Toffana ". Within these vials was a fluid which was supposed to be for cosmetic purposes. It was actually a concentrated solution of arsenious acid. Toffana carried out her nefarious trade from girlhood until she was nearly seventy years of age, and during this time she was instrumental in the poisoning of nearly six hundred people. The reasons for her non-apprehension for so long a time were, first, that she would deal only with individuals after due safeguards had been built up; secondly, she frequently changed her abode and adopted many disguises, and, finally, in an age in which it was rife, she made great show with superstition and religion. The Popes Pius III and Clement XIV are said to have fallen victims to her miraculous oil which was said to ooze from the tomb of St. Nicholas. When eventually the sale of Aqua Toffana was traced to her she took refuge in a convent, from which the abbess and archbishop refused to give her up, and from there she continued to sell the aqua for a further period of twenty years. " Aqua " was a suitable designation, for it was odourless, tasteless and colourless. Eventually public indignation was roused to such a pitch that the convent was broken into by a body of soldiers and she was handed over to the authorities. She was subsequently tortured until she confessed in 1709, and then, after she had been strangled, her body was thrown into the garden of the convent which had sheltered her.

Even as the syphilitic disease had spread from the Neapolitan ports across France in the fifteenth century, so did the epidemic of poisoning spread from Italy to France in the seventeenth century, and it became such a rage that in the reign of Louis XIV a special tribunal was established, the " Chambre Ardente ", for the sole purpose of suppressing and punishing this class of offender. Of the scores of poisoners who were brought to justice by this famous court, none commanded such widespread interest as that of the distinguished aristocrat, Marie-Marguerite d'Aubray, la Marquise de Brinvilliers. We see in her a woman who had every advantage in the way of high birth

PLATE XV

Lucrezia Borgia

and position, great wealth, influential connections, singular beauty, fascinating manners and elegant accomplishments, yet throwing it all to the winds like rain for the mere gratification of carnal passion. Her husband, the Marquis, invited a friend, Captain St. Croix, who was an officer in his regiment, to stay with them. It was the Marquise's father, Lieut. d'Aubray, who was quickly incensed with the knowledge of his daughter's indiscretions with Captain St. Croix and he it was who obtained a " lettre de cachet " and had the Captain sent to the Bastille. Unfortunately, Captain St. Croix there learnt a lot about poisons from an Italian poisoner, Exili, and he divulged the secrets to la Marquise. This " Jezebel ", as she has been described, decided to become a finished actress in the art by visiting the hospitals, particularly the " Hôtel Dieu ", day after day, in the guise of a Sister of Charity, to experiment upon helpless victims. In the course of this diabolical work she managed to produce effects so cleverly that they appeared to be merely an aggravation of the symptoms which she was ostensibly endeavouring to relieve. Her gentility, her sympathy, and her tender compassion, became a byword in the " Hôtel Dieu " and upon her seemingly saintly head untold blessings were invoked by a large number of sufferers, whom she was secretly dispatching to the dead-house. Subsequently, she poisoned her father, her sister and her two brothers because they opposed her amorous intrigue. Unfortunately for her, Captain St. Croix lost his life from the inhalation of deadly fumes in his laboratory. On examining his effects the authorities came across a small box to which was attached a note requesting that after his death it should be dispatched to the Marchioness. On opening the box it was found to contain a large collection of poisons, including corrosive sublimate, antimony and opium. When the Marquise heard of the death of her lover she at once made every endeavour to obtain the box by bribing the officers of justice, but without success. She then fled to Belgium and entered a convent at Liége from which she was subsequently decoyed by an agent disguised as a monk. She was brought back to Paris, tortured into confession and finally burnt near Notre-Dame in July, 1676.

> Oh, you do bear a poison in your mind that would
> not let you rest in paradise.

Even a brief account of poisoners in these times would be incomplete without some description of the hateful Borgias [see Plate XV]. They were of Spanish origin and came to Italy in the time of Pope Calixtus III about the year 1455. Rodrigo was the first to become notorious. He was born in 1431 and after serving for some time as a soldier, he entered the priesthood, and somehow rose to be head of the Church as Pope Alexander VI. He had five children by his mistress, Vanozza de Cattanei; of whom *Cesare*, afterwards Duke of *Valentinois*, and *Lucrezia*, (who became the Duchess of Ferrara) were notorious.

When Rodrigo became Pope Alexander VI he bestowed honours and titles on all his children, with the exception of Cesare, whom he had specially reserved for the purple. In order to do this he had first to remove the stain of his birth and he did so most effectively in a bull dated October 17th, 1480, declaring that Cesare was the son of Vanozza and Dominico d'Arignano. Later, in 1498, another youth called " Romano" was introduced to the household and the pope, declaring him to be Cesare's son, created him Duke of Nepi. The Pope himself was, however, Romano's real father, but his mother remains unknown.

Cesare's carrer of crime seems to have come to the fore on account of his jealousy over his sister Lucrezia. The latter's first marriage had been dissolved by the Pope and she then married Alfonso, Duke of Bisceglie, the natural son of Alfonso II, King of Naples. Soon after the birth of their first child the Duke was attacked by ruffians and severely wounded. His liquidation is described graphically by the chronicler as follows:

" On the night of July 15th, 1500, on which solemn ceremonies were taking place to celebrate the jubilee of the Pope, a young man staggered into the pontifical apartments endeavouring to stem with his hands a stream of blood which gushed from a large

wound in his chest. It was the Duke of Bisceglie, Alfonso of Aragon, Lucrezia's second husband. Consternation was caused when it was spread abroad that a band of assassins in the pay of Cesare had attempted to assassinate him near the steps of St. Peter's when on his way to the celebration. The young man, who is said to have been of a kind and gentle nature, fell at the feet of the Pope. Lucrezia, and his sister, Sanzia, who were standing by, both fainted away and were carried into a room of the tower behind the Pope's chambers. He is said to have been nursed by the two women and to have nearly recovered when one night, in Lucrezia's absence, he was strangled with a cord in bed under the eyes of Cesare."

Some time later Cesare became violently jealous of Pedro Caldeson, who was an attendant in his sister's household. In a raging passion he pursued the man right into the pontifical apartments and assassinated him in the presence of the Pope. " Even so ", says the chronicler, " that the pontifical garments were splashed with blood."

The number of deaths from poisoning attributed to the Borgias is astounding, and one has to realize that, at that time, poison had become the standard weapon in the social and political life of the country. In both political and ecclesiastic circles it procured office by removing rivals and generally smoothed the way, and made life less bothersome.

The picture painted by Apollinaire may not be quite correct, but it is accurate and lurid enough to be recorded. It is an account of a fête held in the Vineyard of St. Peter-in-Chains:

" La Vanozza receives the cardinals and the ambassadors, and, after being introduced to one another, the guests disperse about the vineyard and exchange conversation and courtesies. Later she disappears and joins Cesare in a room on the first floor of the building. She finds him with his sleeves rolled up, bent over a kneading-trough, and absorbed in his task. This room was reserved for Vanozza and Cesare only the Pope shared with them the right of entry and no one else was allowed to cross the threshold. On the floor lay several large, shallow copper dishes, some of which were entirely covered with verdigris and from which a colourless-looking liquid was being evaporated. One of these dishes was always placed nearer the fire in order that the heat might hasten the evaporation.

" As La Vanozza enters, Cesare remarks: 'Yet I forbade you to make a fire.' ' I only put a few live coals on to hasten the result ', she replies, ' I did not make enough for it to be possible for the powder to scorch; if I had not done it we should not have had the powder to-day! '

" ' It is not so much for fear of it scorching, but because of the cinders which mix with the powder and render it less fine ', says Cesare. ' Happily, Cardinal di Riaro is short-sighted! This is quite enough for him in any case, but for the others—hand me the tart dish. It should be dry by now.'

" La Vanozza lifts the heavy, red copper dish by the two handles, and on it may be noticed a mouldiness or greenish spots caused by a settling deposit. With a hare's paw Cesare collects this powder, then, with an ivory knife, he carefully scrapes the copper and mixes the residue in a marble mortar. From it he takes in small pinches some of the powder and places it in another mortar of agate and reduces it in a pestle to an impalpable dust until it is like a morsel of polished silver.

" ' Give me the manna ', says Cesare. La Vanozza hands him the arsenic which he calls by this name, and he mixes some with the powder in the mortar, passing the mixture again under the pestle until thoroughly incorporated, and then, his task completed, he stands erect and exclaims: ' God said: " Let there be light ", and there was light. We Borgias can say: " Let there be night ", and there shall be night '. He then remarks that it is time for luncheon. La Vanozza leaves him and retraces her way. When she is gone, the copper dish being empty, he voids urine in it to replace that which has evaporated and the salts of which he had just utilized. The salts which resulted, combined with the verdigris, were then mixed with arsenic, and this formed the famous poison which the Borgias called ' La Cantarella '. That which the Borgias utilized in conjunction with arsenic without knowing it, was phosphorus, a secret which had been

divulged to the Borgias by a Spanish monk who also knew the antidote for it as well as an antidote for arsenic; one sees, therefore, that they were well armed."

Alexander VI commenced a series of his horrible executions by the murder of Djem, the son of Mahomet II. The latter had been captured by the cavaliers of Rhodes and handed over to the Pope. Some little time after this, Charles VIII, King of France, demanded that Djem should be given over to him as a hostage in order that he might gain a more efficacious control over Turkey. But the wily Sultan had foreseen this move and had previously arranged a little sum of 300,000 ducats with the Pope. The result of this arrangement was that Djem died as soon as he had been handed over to the French from poison which had previously been given in his food.

As the natural heir to the cardinals, Alexander allowed them to become rich from their administrations, and when he considered that the time was ripe, " La Cantarella " brought about their speedy dispatch. It is stated in the chronicles that the cardinals of Modène, Mechiel and Arragon were thus poisoned in succession, and the Cardinal Orsini, convicted of a plot against the Pope, was duly imprisoned. A few days later he, too, died of poison and it is stated that he had been given the Borgias' famous slow poison called " Venenum Attemperatum ". Finally, the cardinal's entire family were dispossessed, and the men were killed, while the women and children were poisoned.

One could go on recounting the crimes of the Borgias almost indefinitely, but you will be more interested in their respective fates. Alas, I regret to tell you that, after many vicissitudes, Cesare died in battle. As for the Pope, some chroniclers insist that he died of a fever within a day or two. Others maintain that he was a victim of his own designs. It is said that he endeavoured to dispatch the very wealthy cardinal, Datary Arian de Corneto, by poisoning some sweetmeats. The latter, however, was wily enough to offer 10,000 ducats in gold to the Pope's carver, who then agreed so to arrange the sweetmeats that the Pope himself was poisoned. According to yet another story, the Pope had invited nine cardinals to dinner hoping to dispatch them all with some poisoned wine. Unfortunately, he entered with Cesare earlier than the time fixed for the rendezvous and asked for a glass of wine for himself and Cesare. This was served by a junior servant who, knowing nothing of the plot, gave them the poisoned wine. The Pope died and Cesare recovered after a serious illness.

Both Burchard and Voltaire deny these stories and state that the weight of evidence suggests that the Pope died of a malignant fever. Whatever the cause, it seems certain that his body swelled enormously after death and that there was early severe putrefaction.

The Case of Elizabeth Fenning

On April 11th, 1815, Elizabeth Fenning, aged twenty, was tried at the Old Bailey for having attempted to poison her employer, a Mr. Olibar Turner, his wife and his father. Elizabeth had made some dumplings for dinner and after eating them, all the family (as well as the maid herself and a young apprentice in the house) suffered from violent pains and vomiting. Mr. Turner said he suspected that they had all been poisoned with arsenic, and next morning he found white arsenic in the bowl in which the dumplings had been mixed. He then stated that a packet of arsenic labelled " Arsenick, Deadly Poison ", which he left in a drawer in his office, was now missing. He also stated that he had observed that the knives they used in cutting the dumplings had turned black.

Mr. John Marshall, a surgeon who was called in, confirmed the fact that arsenic was present in the pan and that the family had been suffering from arsenical poisoning, from which they had all speedily recovered.

Despite the fact that no trace of arsenic was found in the remaining yeast and flour, the girl was found guilty and sentenced to death. The trial aroused great public interest in London, and before long there was an outburst of popular feeling against the conviction, it being generally held that the evidence was insufficient to prove the girl guilty. Several meetings of influential citizens were held agitating for a remission of

the sentence. The Prince Regent, the Lord Chancellor, and the Secretary of State were duly petitioned in turn, but to no avail.

Elizabeth Fenning, at the age of twenty, was executed at Newgate on July 26th, 1815, exclaiming: " I die innocent, but God will convince you by a circumstance this day." But nothing happened on that day as Elizabeth understood it. The sun descended over the myriad mass of struggling and striving life as he had done through eons of time, *and infinitely far away there were other worlds that shone as stars in the night that was deeper than death.* But, nearly twenty years later, in 1834, her employer, Mr. Olibar Turner, died in a workhouse and confessed before his death that he had put the arsenic into the dumplings and sworn away the girl's life.

We are not told of any motive for this dastardly act, and we are left wondering whether it was the expression of a fiendish revenge for the girl having refused his amorous advances.

According to the old Hebrew word " Yom " a day is a period of time, and if the mills seemed to turn slowly, one can at least imagine something of the searing vicissitudes of life that Mr. Olibar Turner must have known to end his days in the workhouse nearly twenty years later.

I died innocent, but God will convince you by a circumstance this day.

Catherine Wilson and Colchicum

Many are the crimes of passion and many are the crimes of greed. The case of Catherine Wilson is interesting in the latter respect.

In April, 1862, a Mrs. Connell had been separated from her husband and she invited Catherine Wilson to tea, as the latter was obviously one of those understanding women who might effect a reconciliation. Immediately after tea Mrs. Connell was taken ill. Wilson ran out, ostensibly to the nearest chemist's shop, and brought back a bottle of " Black Draught ". When Mrs. Connell complained that it was very hot and burnt her mouth, Wilson said it was because she had warmed it and begged her to take it all. It made her feel very sick and blistered her mouth. Mrs. Connell also noticed that even the bedclothes were burnt where a few drops of the liquid were spilt.

She decided to inform the police, but Wilson had suddenly disappeared. As luck would have it, however, some time afterwards a policeman met and recognized her in the street. She was subsequently tried, but the jury considered the evidence insufficient and she was accordingly discharged. Meanwhile, the C.I.D. had been making inquiries and they found that wherever Wilson had lived there had been mysterious deaths. It transpired that in 1853 she had been living as housekeeper to Captain Peter Mawer, a master-mariner of Boston, who took colchicum for his gout. He died suddenly in 1854 and bequeathed all his property to Catherine Wilson.

Two years later Wilson was living with a man called Dixon in the house of a Mrs. Soames of Bedford Square. Dixon died suddenly in mysterious circumstances but Wilson remained, being on friendly terms with Mrs. Soames. About Christmas, 1855, Mrs. Soames inherited some money from a half-brother. Not long afterwards she was taken ill with attacks of vomiting and became so weak that a doctor was called in. Wilson nursed her with unswerving devotion and even took charge of the medicine and administered every dose herself. Despite her constant care (or because of it) Mrs. Soames died in agony a few days later. The doctor at first refused to give a death certificate, but, after the autopsy, he certified that death was due to natural causes.

At the trial Dr. Taylor deposed that all Mrs. Soames's symptoms pointed to colchicum having been administered in large and repeated doses. The same symptoms were now recognized as having been present in the case of Mr. Dixon, who had been living with Wilson in Mrs. Soames's house.

Despite the fact that there was no proof that Wilson had either obtained possession of or administered colchicum, she was found guilty and sentenced to death. Soon afterwards it was found that a Mrs. Jackson, with whom Wilson had lived on friendly

terms at Boston, had died after four days' illness with similar symptoms and that she had £120 in her possession before her death. Her body was exhumed in 1860, but no trace of colchicum or other poison was detected in the stomach. Nor was this the end, for it was subsequently discovered that a Mrs. Atkinson, a milliner of Kirkby Lonsdale (who always stayed with Wilson whenever she came to London to make business purchases) suddenly died when she was with her in 1860. When her husband arrived in response to a telegram he asked what had become of the £100 in notes which his wife had sewn in her clothes for the purchases, but he was happily reassured by Wilson who explained to him that Mrs. Atkinson had told her that she had been robbed at Rugby on her way to London. Mrs. Atkinson's body was exhumed in May 1852, but again no trace of poison was discovered. Catherine Wilson was thus responsible for five deaths with colchicum. Had she succeeded—as she almost did—with the sixth, it seems unlikely that her career of crime would ever have been revealed. Her fiendish cunning was such that she appeared always in the guise of a ministering angel. It requires but little imagination to see her holding the hand of each of her victims and gazing into their eyes with tender light, whilst they in turn paused in their agony to express their heartfelt gratitude, almost with their last breath, for such devoted nursing, so seemingly devoid of self.

It was in keeping with her character that she neither made any confession nor showed the slightest compunction for her crimes. At least twenty thousand people were present at her execution and she faced them all without a trace of emotion on her face, apparently as indifferent to them as she was at the thought of her cold-blooded and fiendish murders.

The Bravo Case

I shall now recount for you the strange mystery of the Bravo case. Mr. Bravo, a young barrister, married a wealthy and attractive young widow, a Mrs. Ricardo. It was known that they did not agree very well, particularly as Mrs. Ricardo had informed her husband of a former lover, and this rankled continually in his mind. On Tuesday, April 18th, 1876, after breakfast at their home at Balham, Mr. Bravo drove his wife into town, and on their way a very unpleasant altercation ensued. After arriving in town, Mr. Bravo spent a normal day, had lunch at a restaurant with one of his wife's relatives, and later walked to Victoria Station with a friend and fellow barrister, whom he invited for dinner on the morrow. On arriving home at 4.30 he went for a ride, but the horse bolted and carried him a long distance, so he arrived home tired and exhausted. At 6.30 he was observed to be leaning forward in his chair looking ill. He ordered a hot bath and when getting into it he put his hand to his side and cried aloud with pain. He seemed ill all through dinner, but this does not appear to have been noticed by his wife or her companion, a Mrs. Cox. All partook of much the same food at dinner, but Mr. Bravo was the only one who drank burgundy, the others having sherry and marsala. The butler had decanted the burgundy for some time before dinner was served. The wine was kept in a cellaret in the dining-room and before Mr. Bravo's illness it had been opened once by Mrs. Cox, his wife's companion and once by the maid, and it was opened subsequently by Mrs. Cox who went to fetch some wine for Mrs. Bravo.

At 8 o'clock the party adjourned to the morning room and retired to bed at 9, the ladies to their rooms, Mr. Bravo to his. After the maid had taken wine and hot water to her mistress's bedroom at 9.30 she met her master at the foot of the stairs and thought he looked queer and strange in the face, but he did not appear to be in pain. He usually spoke to her, but on this occasion he merely looked at her twice and passed on. Mr. Bravo, according to Mrs. Cox, then entered his wife's dressing-room and spoke to her in French with reference to the wine. This had frequently been the subject of unpleasant remarks, but when questioned about this particular conversation, Mrs. Bravo said she had no recollection of it.

After this discussion on the wine, Mr. Bravo, Mrs. Bravo and Mrs. Cox all retired

to their respective bedrooms. A quarter of an hour later Mr. Bravo opened his door and shouted to his wife: "Florence! Florence! Hot water!" The maid then ran into Mrs. Bravo's room calling out that Mr. Bravo was ill. Mrs. Cox, who had not yet undressed, ran at once into his room and found him standing at the open window and apparently vomiting. She later averred that Mr. Bravo said to her: "I have taken poison. Don't tell Florence." After further severe vomiting Mr. Bravo sank to the floor unconscious and remained so for some hours. A doctor had been sent for, but as soon as Mrs. Bravo arrived on the scene she was very excited and insisted on another doctor being sent for who lived nearby. Both doctors duly arrived and endeavoured to get him to swallow restoratives, but he was unable to do so. Then, as his condition was very critical, they sent for Dr. George Johnson of King's College Hospital. He concluded that some irritant poison had been taken. At about 3 a.m. the patient recovered consciousness and was asked at once: "What have you taken?" But he declared most solemnly and persistently that he had taken nothing except some laudanum for toothache. He remained conscious until he died fifty-five and a half hours after the onset of his illness. His room was then searched, but no trace of poison could be found. There was only the laudanum and chloroform for toothache, and it is noteworthy that Mrs. Cox, the companion, told one of the doctors that she thought he had poisoned himself with chloroform. Autopsy showed all the signs of a severe irritant poison. All the vomit had been thrown away, but, fortunately, some vomit had remained on the leads of the house beneath the bedroom window, though the rain had washed most of it away. That which remained was collected and handed to Professor Redwood, who found a large amount of antimony. This poison was also found in the liver and other parts of the body, and it was concluded that altogether nearly forty grains had been swallowed.

Despite the most careful investigation and inquiries the possession of antimony could not be traced to anyone, nor could it be discovered how the poison had been obtained or administered. The Bravo case will probably remain forever, therefore, among the unsolved poison mysteries. There are, however, some points about which we are given no information and which clearly give food for reflection. We are only told that Mr. Bravo was a sane, healthy young man of thirty who was as normal as any young barrister could be. We are given no information with regard to his wife's previous history. How did her first husband die? Why was his body never exhumed? How long had her companion, Mrs. Cox, been associated with her, and what was her previous history? How did her husband die? Was she very devoted to Mrs. Bravo? Why did she tell the doctors that she felt sure that Mr. Bravo had poisoned himself with chloroform, when in reality he had been poisoned with antimony? Why did she say throughout the whole course of the proceedings that Mr. Bravo had confessed to her that he had poisoned himself, whereas in all the hours that remained of his life he stoutly insisted that he had only taken the laudanum for toothache? If he had wanted to poison himself would he have chosen one of the most painful and distressing irritant poisons when he had only to reach out his hand and drink enough of the toothache drops to sink quietly into the arms of Morpheus for ever? For obvious reasons irritant poisons are rarely taken as a means of suicide by enlightened and cultured people! One question, as always, raises another. What was the butler's reputation? Was he, too, in the service of Mrs. Ricardo before she became Mrs. Bravo? Why did Mrs. Bravo take so long to dress and come on the scene when he flung open the door and cried in agony: "Florence! Florence! Hot water!"? You will say that she was excited and insisted on sending for a practitioner near at hand, but, as the record of poison cases shows, that means nothing!

If I have raised these questions it has not been with the idea of casting suspicion on anyone, but merely to show the logical and persistently thorough way in which the inquiry should proceed in such cases.

(*Part II will appear in the next issue of this* Journal)

PRACTICAL POINTS IN THE HOME TREATMENT OF TUBERCULOSIS*

BY

C. de W. KITCAT, M.R.C.S., L.R.C.P.

Consultant Chest Physician, South Somerset

In the past most patients with tuberculosis requiring treatment were admitted to a sanatorium. In recent years, however, bed shortage and new drugs have led to treatment being given by general physicians or surgeons and the family doctor. This is especially so in " closed " cases of tuberculosis, such as cervical adenitis, peritonitis, and genito-urinary tuberculosis. However, lack of experience with the chemotherapy of tuberculosis sometimes leads to the incorrect use of the drugs. Most cases of tuberculosis sooner or later come to the notice of the tuberculosis chest physicians and it is not uncommon for them to find that a patient has been given prolonged streptomycin alone, and that this may have made the tubercle bacilli resistant to streptomycin.

My first point, therefore, is a plea for co-operation between all branches of the profession in the treatment of tubercular conditions. I am not for one moment suggesting that the tuberculosis chest physician is the only person who should give treatment. I am, however, very sure that any chest physician would be very glad to co-operate in the planning of treatment, for it seems likely that the amount of the new drugs which can be given to any one patient in his life-time without his tubercle bacilli becoming resistant to them is strictly limited. It is, therefore, of the utmost importance to use judgment and experience in the timing, length and dosage of courses of chemotherapy.

DRUG THERAPY

It is essential to adhere strictly to the Medical Research Council's recommendation that streptomycin, P.A.S. and isoniazid should only be given in combination and never singly.

Streptomycin is given nowadays usually as the sulphate or calcium chloride solution. Injections are given intramuscularly, the commonest site being the upper and outer quadrant of the buttocks. It is important to see that nurses appreciate this, for I have seen considerable sciatic pain resulting from an unfortunately placed injection. The dilution of the drug is also important if injections are to be painless. One gramme of streptomycin will dissolve in 1 c.c. of distilled water but it will be found, however, that 4–5 c.c. of water will be much pleasanter for the patient. A short post-injection massage of the area also helps in this respect. Nurses giving streptomycin should be told to wear rubber gloves, for it is possible to develop a dermatitis from continual contact with the solution and once it has been acquired it can be very troublesome.

The dosage varies; one gramme a day may be given in certain types of case, and with these daily dosages the patient is best in hospital. For cases at home the American custom of giving one gramme twice or three times a week works very well and is less demanding upon the time of busy district nurses. The one gramme is given in one dose for there appears to be no advantage in dividing it. One great advantage of giving streptomycin twice or thrice weekly is that it takes six months or more to give 72 grammes; bed rest is still the sheet-anchor of treatment, and experience shows that it is much easier to keep patients contented in bed while they are having what is to them recognizable treatment. Dihydro-streptomycin has caused deafness and is no longer used.

* A paper read before the South Somerset Medical Club on September 17th, 1953.

It is important to record the evening temperature taken for five minutes in the mouth because toxic manifestations are frequently preceded by a gradual rise of temperature, particularly in the first three weeks of the injections. The commonest toxic developments are rise of temperature, headaches, malaise, and the appearance of a rash resembling either measles or scarlet fever and this has led, on more than one occasion, to a patient being sent into a fever hospital. Should rashes appear, all treatment should be stopped, anti-histamines given and lotion such as lotio calamine oleosum applied.

When the reaction has settled, tests should be carried out to find out whether the patient is hyper-sensitive to streptomycin or P.A.S. or to both. To do this, 0.5 gramme of streptomycin is first given by itself, and, if the patient is hyper-sensitive the symptoms of fever, itching of the skin or return of the rash will occur within a few hours. If there is no reaction the patient is tested with the full one gramme. When this reaction, if it occurs, has settled, the patient is given a reduced dose of P.A.S. such as 2 grammes. If no reaction occurs a full dose of 5 grammes of P.A.S. is given and repeated several times to make quite sure. Some of the patients with fever and rashes can be desensitized by giving minimal and increasing doses of streptomycin and P.A.S., but this procedure is probably better carried out in hospital. Other toxic symptoms are giddiness, deafness and blurring of vision and in these cases streptomycin should be abandoned completely.

Lastly, except when streptomycin is given for a few days and only for infections other than by the tubercle bacillus, it should *never* be given alone and always in combination with P.A.S., or isoniazid. A continued watch too should be kept upon the total number of grammes of streptomycin which have been given in a patient's life and the mistake avoided of giving too much too early. Granted that the combination with another drug, such as P.A.S., postpones the time when the tubercle bacilli will become streptomycin-resistant, it seems likely that by the time a patient has had 200 or so grammes that this may well have occurred and therefore a regular note of the streptomycin score to date is very helpful.

P.A.S. (*para-amino salicylic acid*) should not be given by itself but in combination with streptomycin or isoniazid. The dosage varies between 10 and 20 grammes a day and the Medical Research Council's investigations have shown that the larger dose is more protective against the development of streptomycin resistance than is the smaller one. On the other hand P.A.S. is rather unpleasant and is apt, particularly in women, to cause nausea, vomiting and looseness of the bowels sometimes amounting to severe diarrhoea. In consequence the intermediate doses of 12 or 15 grammes a day of P.A.S. are often employed. The drug can be given in solution, or in cachets, or in enteric coated tablets—the last in the hope that if the P.A.S. is not liberated in the stomach the nausea and vomiting will be prevented. Some patients prefer one form and some another, but all too frequently if the patient is very sensitive to P.A.S. he will not tolerate it in any form. The requisite dose should be given preferably four times a day after food, never on an empty stomach. For abdominal discomfort the N.F. Pulv. Mag. Trisilicate Co. given with or after the dose sometimes helps; opium in the form of *Tinct. Chlorof. et Morphine Co.* or *Tr. Opii.* may offset looseness of the bowels. There are many different proprietary forms of P.A.S. in cachets, tablets, etc., made up in different strengths so the doctor must be careful to prescribe the correct daily dose. In the home treatment of tubercle, P.A.S. is particularly useful in combination of isoniazid because injections are avoided.

Isoniazid (*Iso-nicotinic acid hydrazide or I.N.H.*) is the latest of the chemo-therapeutic products in common use. It should not be employed by itself. Various doses have been used but nowadays the common dose for most cases is 200 milligrammes daily, given as one 50-milligramme tablet four times a day. Various side effects and toxic symptoms such as constipation, difficulty in passing urine, dizziness, hyper-reflexia, twitchings and nervous symptoms have been described but in the dosage suggested they seem to be very rare. Advantages of isoniazid are the rapid development of a feeling of well-being, and an increase in appetite. From the Medical Research Council's investigations it seems that when using streptomycin in combination with

isoniazid to prevent resistance to isoniazid developing, the streptomycin should be given daily and not two or three times a week. In the past it was recommended that isoniazid should be given for one month only but from the latest report of the Medical Research Council in the *B.M.J.* of November 7th, 1953, it now appears that isoniazid can be given for three months provided that it is combined with daily streptomycin or 20 grammes daily of P.A.S. In addition the result combined with P.A.S. over a period of three months appears to be as good as with streptomycin combined with either P.A.S. or isoniazid.

RESULTS

The various combinations of these drugs produce their most striking results on recent and early lesions, and their least effect on cases of long-standing tuberculosis with much fibrosis. In general, too, cases of glandular tuberculosis seem to derive less benefit than lesions in other tissues. In advanced pulmonary tuberculosis, however, is always worth while giving short courses of the drugs as these reduce cough, sputum and fever considerably for a time and also have some psychological effect.

The methods of chemotherapy are not a complete cure of tuberculosis in themselves and bed rest for long periods remains as essential as ever. The majority of cases of pulmonary tuberculosis too, require artificial pneumothorax, pneumoperitoneum or a phrenic crush. Many cases also will require such major operations as thoracoplasty and lung resection. In recent years however chemotherapy and methods of lung relaxation have produced such startling improvement, even in some advanced cases, that major surgery has become possible. As a result of all this the death rate from tuberculosis has declined in a most satisfactory way.

It is of interest that with all the latest methods of treatment available, the patient nowadays spends a longer time in bed and under treatment than he did in previous years, and, whereas in the past six to nine months in a sanatorium was usual, the patient nowadays is often in hospital for one and a half years or even more. On the other hand the results are undoubtedly very much better and we feel that at long last we are really getting to grips with this troublesome and tragic disease.

PREVENTION BY B.C.G. INOCULATION

B.C.G. is a live vaccine from a strain of tubercle bacillus isolated in 1902, which through repeated sub-culture became so attenuated that since 1930 it has never been virulent for man. Though incapable of causing tuberculosis, it causes production of antibodies and converts the tuberculin-negative subject to a tuberculin-positive one. Tuberculin-negative persons are much more likely than tuberculin-positive persons to develop tuberculosis when they come in contact with infectious cases. Millions of children all over the world have been given B.C.G. without harm and where given there has been a striking diminution in the number of cases of such previously fatal conditions as miliary tuberculosis and infectious tubercular meningitis.

In recent years in this country, B.C.G. has been given to tuberculin-negative medical students and nurses and it is recommended for the tuberculin-negative children of families where there has been a case of pulmonary tuberculosis. The newly born child of a tuberculous mother is removed at once and given B.C.G. and retained in a nursery until it has become tuberculin-positive—a matter of 6–8 weeks. When an infectious adult is removed to a sanatorium any children are left alone for a period of six weeks to give time for possible conversion from recent infection; the child is then tuberculin tested and if negative he is given B.C.G. Where the adult case has never been found to be infectious, B.C.G. inoculation of children can be carried out while they remain in the house with the adult case. To prevent unnecessary needle-pricks for the child, it is common to use tuberculin jelly for the first test and if this is negative to do a final 1-100 intradermal Mantoux before inoculation. Incidentally where B.C.G. has been given to a tuberculin-positive and active case the harm is usually limited to an unpleasant local reaction at the site of injection. In the normal tuberculin-negative case

no malaise or general reaction results and after three weeks a bluish-red papule with an indurated base begins to appear. The local reaction reaches maturity about the sixth week after which it slowly regresses leaving no scar. Occasionally a small area in the centre of the papule may discharge serum or form a shallow ulcer but these heal rapidly with the application of P.A.S. powder.

The conversion to tuberculin positive lasts for about five years but if the child or adult gets occasional doses of tubercle bacilli from natural sources they may remain tuberculin positive indefinitely. However, it is usual to re-test the child annually and to consider revaccinating should reversion have occurred.

There would seem to be a strong case for giving B.C.G. to all tuberculin-negative school-leavers to protect them from tubercle in the 15 to 25 age group when they are starting their life's work, for it is then that they otherwise have the greatest risk of becoming infected.

THE TROPICS ON OUR DOORSTEP*

BY

SIR GEORGE R. McROBERT, *C.I.E.*, M.D., F.R.C.P.

At the first international congress on medical education held in London in August 1953 the Royal Society of Tropical Medicine and Hygiene presented a memorandum on the need for instruction in tropical medicine to students intending to practise in temperate climates. When the subject was announced the majority of the audience left the room " for a breather ". What, thought they, could tropical medicine possibly mean to men whose work lay in Northern Europe?

THE RISKS TODAY

Since the New Year the Mark II Comet has flown from London to Khartoum in six hours. It is now possible to leave London in the early morning, to touch down in tropical Africa, to spend some time bathing in delectable limpid freshwater pools, to disport oneself on a sandy beach, to rest under shady trees, to enjoy green salads and raw fruits at a picnic lunch and to return home the same night incubating, among many other infections, the malarias, the dysenteries, typhoid and typhus fevers, relapsing fever, hookworm disease, bilharzial infections of the bladder and bowel, sleeping sickness, loaiasis, onchocerciasis, subcutaneous maggots, and jigger fleas in the soles of the feet—a truly formidable problem in diagnosis to confront a general practitioner in Great Britain where there is no insect-borne disease amongst humans, virtually no protozoal infections, and with worm infestations practically confined to the lumen of the alimentary canal.

The practitioner in this country is increasingly liable to meet with hitherto unexpected problems in diagnosis, for apart from the theoretical much-afflicted tropical tripper to whom I have just referred thousands of visitors come by air each year from India, Africa and South America, all potentially incubating tropical maladies which, in the past, generally became manifest on the sea voyage to England.

The title of my talk is, I think, an apt one. The tropics are virtually on our doorstep or, at most, next door. The aftermath of the recent war, too, has brought to this country enormous numbers of men and women who contracted one or more tropical diseases in Burma, Africa or the Middle East, and as the correspondence columns of our journals indicate, the recent operations in Korea have let loose on our doctors here, numbers of young National Servicemen who contracted benign tertian malaria in Korea and who produced the first symptoms of it many months after their arrival home. We have, further, a great influx of Africans and West Indians to swell the labouring population of this Welfare State, and every part of our tropical Commonwealth sends a quota of students to universities, inns of court, technical institutes and schools, so raising still higher the amount of tropical disease to be diagnosed in this country.

It is, of course, impossible for me to deal adequately with this enormous subject in the time at my disposal. I propose to deal only with the malarias, amoebiasis, bilharziasis, loaiasis, onchocerciasis and leprosy. You may have noted that in my grim

*A paper given by invitation to the Bristol Medico-Chirurgical Society on 10th February, 1954.

catalogue of possible infections I did not mention smallpox and yellow fever. Against these two virus infections we can give absolute protection. Great care is taken by all airlines in checking in detail the vaccination and inoculation history of each passenger, and these two diseases give rise to little anxiety. The development of a perfect attenuated vaccine of the yellow-fever virus, causing no reaction of any kind, no painful swelling on injection, and giving complete protection within ten days of administration and for six years thereafter against the dreaded yellow jack which used to terrorize all mariners sailing from this and other ports to West Africa and the Spanish Main encourages us to hope that before long the viruses of poliomyelitis and encephalitis will be successfully mastered. I find the daily procession of peoples of all nations, colours, creeds and ages (seven days old to ninety years) attending our daily yellow-fever inoculation clinic most interesting, stimulating and thought provoking.

MALARIA

Malaria in human beings is caused by four species of unicellular animals or protozoa of the genus *Plasmodium*, but from the clinician's point of view there are two types of malaria:

The vital-organ sporulators—malignant malaria;

The surface-capillary sporulators—the benign malarias.

The essential difference between the two types is that in malignant or falciparum malaria the multiplication or sporulation of the parasites within the red blood cells takes place within the capillaries of the vital organs in the depths of the body giving rise to multiple blocking of capillaries with acute infarctive and anoxic phenomena in the brain, pituitary and suprarenals, bowel, heart or kidneys resulting according to the organ mainly affected in deep coma, choleraic diarrhoea, precordial pain and collapse or massive shock. Death may be almost immediate.

In the benign malarias the sporulation takes place in the surface capillaries so that although the benign tertian, ovale and quartan forms of malaria are debilitating and exhausting, they are not acute emergencies and are not immediately dangerous.

RECENT DISCOVERY ABOUT LIFE-CYCLE

I should like at this point to spend a few moments dealing with the life-cycle of the malaria parasites. If you refer to the text-books on your shelves you will find in all but the most recent editions an account of how the female anopheline mosquito injects malaria parasites with her salivary fluid as from a hypodermic syringe and that these minute torpedo-shaped sporozoites enter the red blood cells and multiply: when a sufficient number of red cells are invaded and ruptured symptoms become manifest with rigors, flushes and fever. Fifty years ago Schauddin said that he saw the sporozoites enter red blood cells in a wet preparation under a microscope. He was completely mistaken. The world was misled for half a century. The parasites injected by the mosquito do not enter the corpuscles at all. During the recent war my present colleague Sir Neil Hamilton Fairley working on human volunteers in Australia showed that the blood of Smith and Brown who had been bitten by heavily infected mosquitoes, if withdrawn and subinoculated into Jones and Black would produce malaria in them only if the period between the mosquito biting and the withdrawal into the syringe was less than thirty minutes or more than six days. In other words the sporozoites are present in the circulatory blood for a brief period after the mosquito bite—in the plasma—but must rapidly disappear to some unknown destination from which infective forms emerge and are seen in red blood cells about a week later. Where they had gone no one knew till Henry Shortt and his co-workers discovered the liver cycle of malaria parasites in 1948/49/50, first in monkeys and later in human volunteers in benign tertian and malignant malaria. We now know that the malaria parasites enter and

multiply in the polygonal cells of the liver before invading the red blood cells and that what we now call Shortt's tricycle has three parts:

(1) In the mosquito;
(2) In the liver;
(3) In the red blood cell.

From the points of view of prevention and treatment a very important difference has been discovered between malignant falciparum malaria and the benign malarias, and it is greatly to Hamilton Fairley's credit that he predicted this difference on a theoretical basis before Shortt demonstrated it by experiment. The parasite of malignant malaria visits the liver and leaves completely after a few days. The parasites of benign malarias lodge in the liver and merely send out periodic detachments into the blood.

Malignant malaria has been the No. 1 Killer in the tropics in the past and is still the most important disease in the world and is the chief problem facing hygienists and medical administrators in the tropics. So far as visitors to the tropics are concerned, soldiers, sailors, airmen, commercial travellers and tourists—and in fact in all organized labour forces—the problem may be said to be a vanishing one.

Treatment. Thanks to modern synthetic drugs we have absolute power to prevent the development of malignant malaria as certain synthetics, taken in small doses daily, kill the liver-forms before they can develop to the blood-invading stage and are what is known as causal prophylactics.

Paludrine (Proguanil) is the outstanding example. No European going to the tropics should contract malignant malaria. Blackwater fever *should* be a thing of the past—and very nearly is. We should never see malignant malaria in this country if all persons passing through malarious areas were to take their daily prophylactic pill during the period of risk and for a fortnight thereafter. But tragedies continue to occur—chiefly amongst the occasional or casual traveller passing through Africa from one temperate zone to another, from Britain to Cape Town for example. A mosquito bite in Nigeria may cause an acute seizure, hyperpyrexia, coma and collapse in Bristol or Johannesburg, and unless the practitioner is ready with his syringe all may be over in a very short time.

When acute symptoms develop in one recently returned from the tropics—take a blood slide (a thin and thick film and a thick drop if possible). Have it examined by an expert at leisure but do give a potent antimalarial drug as soon as possible by mouth if the patient can swallow readily—intramuscularly or intravenously if he cannot.

Those of you who have served in the forces are probably familiar with the German antimalaria weapon Atebrin (Mepacrin) which enabled us to defeat the Japanese in South-East Asia. It is still a valuable drug, but its German successor Chloroquin (Nivaquin, Resochin, Aralen) is probably the best drug for the treatment of malaria.

The prophylactic drugs Paludrine (Proguanil) and Daraprim have no place in the treatment of acute malaria in Europeans. They come into action much too slowly.

Please take a blood slide if you can, but don't wait for the result before starting to treat the acute disease.

Benign malaria. I now pass to the benign oft-relapsing malarias of which we are seeing a great deal in Britain today.

A fortnight after leaving the tropics the visitor usually stops taking his suppressive paludrine or daraprim—malignant malaria is ruled out as a future possibility but the liver may contain dormant or smouldering forms of the benign malarias. Many months later an attack of malaria may occur even if no clinical malaria had occurred in the tropics. That is the usual story in the young servicemen who are going down with malaria in Britain today. Some of our patients have been treated for weeks or months with antibiotics, sulphonamides and diaphoretic mixtures, arriving in hospital greatly debilitated and with large spleens. Paludrine or daraprim kept the malaria suppressed, and whilst taking one tablet a day no clinical symptoms will occur.

TREATMENT OF RELAPSES OF BENIGN MALARIA

We now know certain drugs of the 8-amino-quinoline series which do act on the liver forms of the benign malarias. The earlier numbers of the series — the German plasmoquin (pamaquin) were rather toxic in even small doses, but the latest American production Primaquin is notably less toxic and more efficient. Given in small doses for a fortnight it destroys the liver-forms of benign malaria in a high proportion of cases. The American Army has been giving this drug to returning troops on the voyage across the Pacific to the U.S.A., and the incidence of benign tertian malaria is now very low in Korean veterans in the U.S.A. and Canada. Primaquin has recently become available on this side of the Atlantic. It is not suitable for use in general practice—the patient should be under close supervision during the whole course of administration.

AMOEBIASIS

I now pass on to consider briefly the subject of infections with *Entamoeba histolytica* which give much trouble to practitioners in this country. We have more references from general practitioners for consultation at the Hospital for Tropical Diseases for chronic bowel disease than for any other complaint: the history given by the patient is generally that the trouble started on a trip to South Italy, Spain or Greece or, of course, on war service in the Middle or Far East. The subject is not any easy one to deal with—even the parasite itself is not fully understood.

The cause of amoebic dysentery is a freely motile grey protozoon—*Entamoeba histolytica*—with well-defined ectoplasm, rapidly protruded pseudopodia, apparently purposive movement over the microscope field and containing in its protoplasm several red blood cells in process of digestion. It burrows into the submucous coat of the colon —chiefly in the caecum, but in heavy infections extending right down to the lower rectum. In the submucous layer by a process of lysis and gelatinous degeneration characteristic flask-shaped ulcers are formed with narrow ulcerated openings to the surface. Between the ulcers the lining of the colon is surprisingly normal in appearance. The amoebae enter radicles of the portal vein, travel to the liver and may reach the lungs, brain, muscles and other parts of the body causing far-reaching effects.

Entamoeba histolytica can be cultured quite easily on artificial media but a bacterium —such as *B. coli*—must be grown with it. It cannot be grown alone.

Much speculation at present rages with regard to the pathogenicity of *E. histolytica* by itself. Does it require a second organism to enable it to penetrate the gut wall? Do secondary infections determine the severity of the intestinal lesions? It has been generally taught that amoebic dysentery has a mild onset in contra-distinction to the acute start of bacillary dysentery, but one meets cases which start in a fulminating fashion, with high fever, severe constitutional symptoms and sloughing of the gut wall. Many now believe that it is secondary invaders which are responsible for such a picture.

The characteristic four-nucleate cysts of *E. histolytica* may be found in the stools of inhabitants of these islands who have never left these shores—recently an investigation at a school for mental defectives revealed an astonishing percentage of infections. It may be said, however, that the cysts passed by such non-travellers are under 10μ in diameter, whereas cysts in the tropics generally slightly exceed that measurement. It is probable that there exists here and elsewhere a harmless smaller form of *E. histolytica* living in the bowel contents as a harmless commensal and with no invasive tendencies.

Management. What is the practitioner to do if cysts of *E. histolytica* are reported in the stools of a person who has never been abroad? If there are no bowel symptoms and if the patient is not a food handler treatment is unnecessary. If the patient has been abroad one assumes that one is dealing with a pathogenic variety and full treatment at the hands of an expert is indicated.

Drug treatment. The sheet-anchor in the treatment of amoebiasis is emetine and its derivatives. The toxic nature of emetine has been over-emphasized but it is wise to keep the patient in bed whilst administering it. The place of the antibiotics Aureo-

mycin, Terramycin and Fumigillin in the treatment of amoebiasis is not yet fully determined. It is probable that a combination of emetine with fumigillin is the best treatment for amoebic dysentery. It is quite clear that the use of antibiotics alone which, under the influence of high-pressure American salesmanship, have displaced emetine in better-class practice in certain parts of the tropics, is producing a crop of liver and lung abscesses, staphylococcal enteritis and mould infections of the gastro-intestinal tract which we did not see when we relied on emetine.

The invasion of the liver by many amoebae causes acute hepatitis which may proceed to the formation of one or more sterile liver abscesses—the largest of which is usually found in the right lobe of the liver. Since Sir Leonard Rogers introduced emetine therapy in 1910 it has been possible to cause amoebic hepatitis or even amoebic abscesses to resolve without recourse to surgery. If such intervention is necessary simple aspiration is generally sufficient. Open operation—so fatal in the tropics in pre-antibiotic days—is practically never necessary except in left-lobe abscesses pointing in the epigastrium or left hypochondrium. During the past year I have seen six patients who have been subjected to unnecessary open operation for liver abscesses treated by antibiotics without emetine.

Chloroquin. Only very occasionally is *E. histolytica* emetine-resistant, and we now have a very powerful second string in Chloroquin which I have already mentioned as the best synthetic drug for treating acute malaria. Chloroquin is highly concentrated in the liver and lung and deals efficiently with amoebae deposited there. It does not concentrate in the intestinal wall and so is useless for the treatment of the bowel infection.

Amoebae may produce lesions in the lung, brain, and skin, and granulomatous tumours of the bowel may be amoebic and may resolve readily when emetine is given. Lungs have been removed for amoebic abscess thought to be malignant. In a recent number of the *B.M.J.* a surgeon near London reported that he had operated on a tumour in the right iliac fossa suspecting malignancy and with most unfortunate results. The tumour was an amoeboma and the diagnosis was not suspected till amoebae invaded the skin from the colostomy. Let me say that in persons who have returned from the tropics a diagnosis of appendicitis, carcinoma of the colon or rectum should be arrived at only after amoebic infection has been excluded.

SCHISTOSOMIASIS

We are very fortunate that in this country human beings have little to fear from snail-borne disease—an occasional accidental infection with the sheep fluke—*Fasciola hepatica*—and transient skin rashes in bathers in some freshwater reservoirs but nothing more.

Everyone knows that the chief curse of Egypt is bilharzial infection with resulting haematuria, contraction and often carcinoma of the bladder, gross splenomegaly, cirrhosis of the liver and ascites, but it is not so well known that both bladder and bowel varieties produce havoc in Egypt and the Soudan—the male and female worms living in the portal vein, their sharp-spined ova finding their way not only to their natural exits in the bladder and bowel but also wandering in the blood currents to sites in the lung, heart, brain and spinal cord in each of which pathological changes can be set up.

From your zoology days you may remember that the sharp eggs rupture in fresh water, that the motile parasite enters an appropriate snail, invades its liver where it undergoes a complicated series of changes. Infective forms—the free-swimming cercariae—penetrate the skin of a human being exposed to infected water, make their way to the portal veins where they grow, couple and produce eggs.

In Arabia and in Africa, from Egypt in the north to Natal in the south, millions of people are afflicted with schistosomiasis. We must not think of it as a disease of the poor who work knee deep in rice fields and swamps. It is a very real danger to visiting

Europeans. Anything which brings infected water into contact with the skin is potentially dangerous, e.g. an angler's wet fishing line, the brief contact with water whilst picking a fish or a shot duck out of water and, of course, untreated water used for washing or drinking. During the past year I have treated doctors and their wives—and picnic guests of those same doctors—who so far transgressed the rules of tropical hygiene as to indulge in freshwater bathing in an area of Nigeria where freshwater snails abound. At the present time I am treating a very heavy *haematobium* infection in a European who fell out of his canoe whilst duck-shooting and also three schoolchildren who contracted the disease whilst on a flying visit to parents in Africa. Very many parents in the tropics now have their children out during school holidays. Malaria prophylaxis seems to be well carried out but the real and ever-present danger of schistosome infection is not fully grasped. School doctors and general practitioners need to be on the look-out for cases of mild dysuria and slight chronic diarrhoea. Ova may be found in the urine or faeces. Complement fixation tests or skin sensitivity reactions and the presence of an eosinophilia will help in the diagnosis.

The treatment of these infections has to be very thorough. Although much research has been done in the hope of producing satisfactory drugs for oral administration, those on the market do not produce the high rate of cure claimed by the makers. We still rely on intravenous sodium antimony tartrate—originally introduced by Christopherson early in the 'twenties. The treatment is tedious—especially in *S. Mansoni* infections—but the eventual results are good in cases taken early.

IMPORTANT NEMATODES

Now I want briefly to deal with three important nematode worms, carried by insects and living not like the round worm, threadworm and hookworm in the intestinal canal —but in the body tissues. These worms are: *Wuchereria bancrofti—Filaria bancrofti*—carried by the ordinary female *culex* mosquito; *Loa loa*, carried by the mangrove fly—*Chrysops*; *Onchocerca volvulus*, carried by the river fly—the black *Simulium* midge.

Filaria bancrofti lives in the lymphatic system—mainly in the lymphatic glands. It produces an enormous number of young microfilariae which swarm in the superficial capillaries during the hours of darkness ready to be sucked up by mosquitoes. The glands occupied by the adult filariae are apt to undergo periodic inflammatory changes. Recurring filarial lymphangitis is a common clinical finding in all parts of the tropics. The clinician is generally aided by a history of previous similar attacks. Retroperitoneal lymphangitis may closely simulate an acute surgical emergency and it is most important in dealing with people from the tropics to inquire about similar occurrences in the past and to examine for signs of hydrocele and other evidences of bancroftian filariasis.

The condition may pass on to a stage of elephantiasis, but this is very rare except in natives of the tropics who are subject to nightly assault by hordes of heavily infected mosquitoes and to secondary streptococcal invasion through fissures and sores in the skin.

Loa loa infection is not dangerous. The infection is caused by a day-biting fly and the microfilariae are found in the blood in the day-time only. The adult worms live in the subcutaneous tissues, are very active and migratory, and may cross the bulbar conjunctiva from time to time. In former days doctors used to grab the worm with forceps as it crossed the ocular conjunctiva, make an incision and extract the worm. The infection gives rise to skin allergies and to the appearance of transient swellings—Calabar swellings—from a hen's egg to a goose egg in size in various parts of the body —very puzzling to those who are not familiar with the condition. The infection is confined to rain-forest areas of West Africa and is seen not infrequently in visitors from this country.

I must now introduce you to a more serious invader—*Onchocerca volvulus*—conveyed by *Simulium damnosum*—an appropriately named small black midge. In

chronically infected persons large cystic easily-shelled-out swellings appear in the subcutaneous tissues, over bony prominences and round joints. They may reach the size of a small coconut.

The cysts contain coiled-up male and female worms. The microfilariae are not found in the blood but in the deeper layers of the skin. In order to establish a diagnosis fine skin shavings are taken and the living microfilariae are seen by the microscope.

The oustanding importance of this worm is the liability to severe eye complications. All the eye tissues are liable to damage. One may get conjunctivitis, keratitis, uveitis and choroido-retinitis often with irreversible changes and blindness. Not without reason has this worm been called *Filaria caecutiens* (the blinding worm) in Guatemala, for in Central Africa and Central America the amount of blindness and visual disability caused by the infection is serious. Europeans are readily attacked. I find that British ladies with their addiction to gardening are singularly careless about applying insect repellents before emerging from their fly-proof houses, thus falling victim to this vicious fly.

It has very recently been realized that the old description of onchocerciasis as " filaria with knobs on " is no longer correct and that the eye may be invaded early by the young of paired but still wandering worms which have not yet settled down in their cystic abodes. Last year I had under my care a European school-child with severe keratitis but with no sign of adult worms. At the present time I have a European lady patient with advanced eye changes, with microfilariae in the anterior chamber and corneae but without any sign of adult worms.

It is therefore incumbent on the practitioner who comes across such eye changes in a recent visitor to or resident in the tropics of Africa and Central America to consider the possibility of onchocerciasis and to refer the patient to an expert for examination by the slit lamp and corneal microscope. Before the eye changes come on, however, there are usaully severe signs of skin irritation or lichenoid eruptions.

Treatment. Thanks to American research during the war we have in diethylcarbamazine (hetrazan-banocide) a powerful weapon against *bancrofti* and *loa* infections and a considerable aid in dealing with onchocerciasis. I would however plead for admission of such cases to a tropical-diseases hospital for treatment as a very cautious approach must be made and cortisone must be available for local and general use at the slightest sign of acute reaction in the eye when treatment is begun.

The British Empire Society for the Blind has recently given £40,000 for the relief of onchocerciasis in West Africa.

The use of D.D.T. in small rivers and streams is rapidly controlling the extent of the river-fly pest in certain places. At Jinga in Uganda—hitherto one of the worst areas in the world for this disease—intensive anti river-fly activity has been in operation for some time so that we now feel that it is safe for Her Majesty to go there in the near future to visit the great new dam.

LEPROSY

I must conclude with some remarks about leprosy. So far as we know no case of leprosy has developed in this country for many years though there are numerous imported cases. I am not sure that such a happy state of affairs is likely to persist. Until recently leprosy was not a notifiable disease. It was realized that changed political conditions in India would lead to the emigration of Eurasians or Anglo-Indians in considerable numbers and that some of these would have leprosy. There are about 200 known cases in this country—how many there are unrecognized we cannot tell.

Now leprosy cases are notified in strict secrecy to the Ministry of Health in London, and after a visit from an expert the local Medical Officer of Health is informed verbally and strictly confidentially by him. But he can do nothing about it. The waiter may go on waiting, the school-teacher may teach infants, and the mother may go on producing children and probably infecting them without let or hindrance. The Minister

of Health has no reserve powers of compulsory segregation of infective cases. It is true that one leprosy hospital exists at Reigate, Surrey—a branch of the London Hospital for Tropical Diseases with accommodation for twenty cases—shortly to be expanded to twenty-six. There is also St. Giles' home in Essex cared for by a band of nuns. But no one can be compelled to go there.

A high porportion of leprosy patients in this country are infective but it must be realized that for the spread of the disease to occur prolonged and intimate contact—as from mother to child—is usually necessary, and that most patients acquire their disease in infancy—but not always. We have three European patients at present who did not visit the tropics till adult life.

In persons returning from the tropics the diagnosis of leprosy should always be kept in mind in connection with complaints of numbness, tingling or unusual rashes and above all in areas of depigmentation with anaesthesia. In no circumstances should the word leprosy be mentioned to the patient for the social implications are so severe. The patient should be referred to an expert (the Ministry will always advise) without his knowing the nature of the suspected illness. A skin biopsy will generally be required to determine the diagnosis—and prognosis. The patient should be completely stripped. We have at the present time a European Military Officer, a British soldier, an Anglo-Indian typist and a Maltese school-teacher who look completely normal in every way when fully dressed; their lesions are all hidden by their clothing.

Despite what you read in newspaper propaganda, leprosy is a very difficult and indeed dangerous disease to treat in Europeans, Anglo-Indians or other half-castes. Modern sulphone therapy is beset with difficulties; it is quite easy to produce a severe or even fatal reaction in the nerves with quite a small dose of sulphone preparation with high fever, acute strangulation of the nerve fibres with complete paralysis and dangerous eye changes.

Many contretemps are met in the course of treatment which at best is of several years' duration—great patience is needed by both patient and doctor.

CLOSING REMARKS

I have no time to deal with the short fevers, such as dengue and sandfly fevers which may now appear here with fast air transport—the vectors are not found here and they will not spread. The possibility of importing bubonic plague by a passenger from Bombay is well recognized and is one reason for surveillance of an M.O.H. of any illness occurring within a fortnight of arrival in this country.

The British Commonwealth and Empire is composed of an aggregate of free and equal peoples but, as in *Animal Farm*, some are more equal than others. The most important inequality which concerns us is that alone among the members of the Commonwealth Great Britain has no powers of exclusion. The introduction of such powers to enable us to shut out criminal and infected persons from other parts of the Commonwealth would, as the Home Secretary said the other day, offend against the sacred principle of the door open for all Her Majesty's subjects. We must therefore continue to expect an inflow of leprosy and other tropical diseases and to be prepared to diagnose and treat them.

COLONIAL MEDICAL RESEARCH

BY

H. S. L. HELLER, M.D., Ph.D., M.R.C.P.
Professor of Pharmacology, University of Bristol

Most medical practitioners in this country know little about the research sponsored by the Colonial Office. Some of this work has distinct bearings on medical and economic problems of countries in the temperate zone. Therefore it may not be out of place to say a few words about this aspect of British Colonial stewardship.

The central organizing body is the Colonial Medical Research Committee (C.M.R.C.). It is aided by regional organizations in some of the Colonial territories, in East Africa by the East African Bureau of Research in Medicine and Hygiene and the East African Standing Advisory Committee for Medical Research, and in West Africa by a similar Standing Advisory Committee which will soon be enlarged to a West African Council for Medical Research.

The C.M.R.C. administers funds allocated to it under the 1945 and 1950 Colonial Development and Welfare Acts. Up to 1953 £12,200,000 has been provided, 14.2 per cent of which has been allotted to medical research directly, and a further 27.1 per cent to work closely linked with medical problems like tsetse and trypanomiasis research. The way in which these monies are used varies widely: research units are maintained in the Colonies, assistance is given to research programmes undertaken by the Medical Departments of Colonial territories, the training of young medical workers is subsidized, special grants are made to research institutions or university departments in the United Kingdom working on problems of interest to the Colonies. A new development which the C.M.R.C. seeks increasingly to foster is the provision in Colonial centres of facilities for senior research workers from British universities for the short-term study of specific problems.

The forms of research work done in the Colonies include medical surveys, clinico-pathological studies and therapeutic trials; infectious and parasitic diseases, nutritional deficiency and endocrine diseases, and physiological problems are studied.

The physiological research is by no means an academic extravagance; it is necessary to provide a basis for clinical work. Thus one of the difficulties of investigating a pathological condition in an African territory is the lack of normal or control data. Thanks to the labour of several generations of physiologists and clinical pathologists, a standard text-book usually contains reliable ranges for, say, the level of most blood or plasma constituents in Europeans or North Americans of most age-groups. Similarly there are numerous investigations giving the results of function tests on various organs of healthy white people. However, very few such facts are known about the inhabitants of Colonial territories. It is, of course, not to be assumed *a priori* that differences will be found, and, if found, that they are due to different racial characteristics. They may be due to the different climatic conditions or the local diets. But whether such differences can be found or not, it is clear that the investigator is not entitled to interpret findings in any pathological condition without data on a representative sample of healthy subjects of the same population. To give an example: a small group of workers from the Department of Pharmacology, Bristol, recently went out to Kampala, Uganda, to collect data on the water metabolism and the renal function of infants and adults suffering from nutritional oedema and liver disease. When we came to apply simple renal function tests like measurements of the ability of the kidneys to

dilute or concentrate the urine, we realized that there was no information on Africans in Uganda or any other part of that continent. Similarly, there have been very few estimations of plasma sodium and potassium levels in Africans. Therefore healthy infants and adults had first to be investigated. But this was by no means easy. There were curious difficulties. For although Africans are not averse to injections (in fact they like them and expect much from them), the *removal* of body fluids is a different matter. This is frequently resented, probably because the African fears that the material will be used for magical purposes. However, when matters are carefully explained to them, it is usually possible to obtain the consent of the African patient or the patient's mother to remove small samples of blood. Assuming then that a number of volunteers have been assembled, will the results obtained be acceptable? Will they really yield " normal " values? It must be remembered that the incidence of parasitic or infectious diseases in many tropical areas is very high even amongst subjects who are apparently well enough. In Uganda, for instance, malaria, hook-worm infestation and amoebiasis have to be thought of. Moreover, nutritional deficiencies are widespread. Gross cases will be admitted to hospital but the majority of the " controls " available may be suffering from the same condition to a minor degree. A statement of Davies and Trowell (cited from Brock and Autret) bears this out. They state that in Kampala there is hardly an adult African liver which does not show fibrosis to some degree, at least when examined microscopically. Such difficulties as these lead to a better appreciation of the work of the pioneer physiologists, done at centres like Makerere Medical College, Kampala, Uganda, the Hot Climate Physiology Research Unit at Oshodi, Nigeria, the M.R.C. Laboratories at Fajara, Gambia and others.

Besides this physiological and biochemical research there are epidemiological and nutritional surveys which will mould future clinical work. The East African Medical Survey not only studies the incidence of diseases like tuberculosis, malaria, relapsing fever and schistosomiasis but also chooses areas for experimental vaccination and for therapeutic trials. Two other examples of recent surveys are an investigation of the epidemiology of human trypanosomiasis in the Gambia, in the course of which over 55,000 persons were examined, and an inquiry into the incidence of endemic goitre in Nigeria. Such surveys may have their peculiar difficulties. For instance, when the victims of filariasis are asked to come to the hospital at night they are reluctant to do so because lions and other wild beasts make their journey hazardous. Nor is the vampire-like activity of research workers who wish to visit infested villages to make blood films at night particularly appreciated.

There are closely related projects designed to identify potential insect and other vectors of diseases, to find their habitat and breeding habits and to learn about seasonal incidence. Much of this work is done in collaboration with entomologists. Astonishing results (astonishing at least to a medical man who knows little about tropical diseases) are sometimes obtained: it was found in Borneo, for instance, where malaria is hyperendemic, that the previously accepted vector *Anopheles maculatus* is almost certainly harmless, and that the chief vector throughout the land is another mosquito, *A. leucosphyrus*, which is not " house-haunting " but breeds in jungle-covered hill ravines. Conventional anti-mosquito measures like the oiling of pools, spraying of buildings with D.D.T. and drainage will therefore not prevent Borneo malaria, but clearance of scrub, sufficient to admit sunlight, results in quick eradication of *A. leucosphyrus* breeding. It would take too long to mention similar research programmes in any detail. Mere quotation of some subjects and sites of field work may be sufficient to show the variety and wide territorial spread of inquiries in progress:

East Africa: distribution of mosquitoes transmitting filariasis; resting habits and physiology of malaria mosquitoes; relapsing fever research (Kikuyu Reserve, Kenya, and Usambara highlands, Tanganyika). Study of snails on Ukara Island in a search of schistosoma species; isolation of polyomyelitis virus (by the Virus Research Institute, Entebbe).

West Africa: study of transmission of filarial infection in the rain-forests and grasslands of the British Cameroons; interrelation between helminthic infestation and

nutrition (Gambia); the distribution of transmitters of the guinea-worm (Nigeria); malaria in pregnancy; study of yellow fever epidemics and isolation of strains (Virus Research Institute, Lagos).

Malaya: study of vectors of filarial infections: malaria control in rural areas; investigation of the animal reservoirs and vectors of yellow fever, dengue, encephalitis and leptospirosis (by the Colonial Office Scrub-typhus Unit in collaboration with a unit from the U.S. Army Medical Service).

Trinidad: entomological research on malaria mosquitoes and the effect of D.D.T. and gammexane. Investigations such as these are frequently combined with therapeutical trials which are very necessary since well-known and accepted drugs may have to be tried on newly discovered strains or newly discovered drugs may have to be tested.

While much time and money is spent on " tropical " infections, increasing attention is being paid to the study of nutritional deficiency conditions. The prevalence of these diseases in many tropical countries has only been recognized in recent times. This is to some degree a result of medical experience in European countries during and after the two great wars. Deficiencies of subsidiary food factors, i.e. vitamins, could be studied in peace-time, but it is mainly under war-time conditions that gross under-nutrition and lack of one or more of the chief food constituents (proteins, fats) occur. These circumstances led to the intensive study and better understanding of starvation oedema and nutritional liver damage, and to the recognition of a peculiar syndrome in infants fed on diets poor in proteins but rich in carbohydrates, termed " Mehlnaehrschaeden " by German investigators. Similar conditions known as " Kwashiorkor " were subsequently reported from East and South Africa. Depigmentation of skin and hair, and dermatoses seem to be more prominent in the tropics than in Europe. However, when, at a recent occasion, the opportunity arose to compare African infants with kwashiorkor with Italian children of the same age-group suffering from nutritional protein deficiency, little doubt was left in the writer's mind about the essential identity of the two conditions. Lack of protein is now generally agreed to be the main cause of this syndrome although regional differences in the symptomology make it likely that additional factors are involved: the amino-acid composition of the protein in the plants used as staple food in various regions may differ, there may be subsidiary deficiencies of one or several vitamins, toxic substances may be contained in the vegetable diets fed, or there may be differences in the proportions of the main food ingredients (carbohydrate/protein ratio). The clinical picture remains nevertheless sufficiently clear to have led to the recognition of kwashiorkor in areas as widely dispersed as East and West Africa, the Belgian Congo, South Africa, the Philippines, the West Indies and Mexico.

It seems likely that adult forms of kwashiorkor exist, both in an acute form frequently seen in Southern India (which according to a personal communication from Dr. C. Gopalan of the Nutritional Laboratories at Koonoor closely resembles kwashiorkor in weanling infants) and in a chronic form seen by us in Uganda in young adults (these patients are usually admitted with liver cirrhosis, oedema and ascites.

During six weeks at the Mulago Hospital, Kampala, we saw twenty infants suffering from severe kwashiorkor. The high incidence and fatality of this disease raises major problems of prevention and therapy in Uganda and many other tropical countries. Both problems are complicated. The main therapeutic task would appear to consist in administering sufficient amounts of proteins yielding mixtures of amino-acids of high biological value. Milk seems the simplest answer, but it has not proved to be entirely satisfactory. Moreover, the amounts of milk needed would be very considerable and in East Africa for instance—where the milk yield of the native cattle is very poor—such treatment would be too costly. A special M.R.C. unit is being organized in Kampala (with the help of the Colonial M.R.C. and the Government of Uganda), to study, amongst other nutritional problems, the use of more easily available plant proteins in the treatment and in the prevention of kwashiorkor. However, measures other than dietary may have to be used for treatment, since a number of children die in spite of prompt protein administration. More rapid reduction of the oedema may

be necessary in some patients; but such treatment presupposes greater knowledge of the causal factors, and of renal function in this disease. It was with this aspect of kwashiorkor that the group of workers from Bristol University were concerned.

Prevention of kwashiorkor poses, if anything, an even more difficult problem than its therapy. Firstly, a very large number of people has to be protected; secondly, purchase of the right type of food is usually beyond the means of an African family; thirdly, the food habits of some tribes would have to be changed. The problem of combatting kwashiorkor is therefore not only medical, it is economic and sociological as well.

Sufficient has been said to convey an impression of the scope and diversity of medical research in British Colonial territories; and if, as many believe, world peace and security depend on the improvement of living conditions in the most needful areas, the general importance of much of this work will be easily recognized.

I am much indebted to Dr. R. Lewthwaite, O.B.E., F.R.C.P., Director of Medical Research, Colonial Office, for reading this article before its publication.

A RADIOLOGIST VISITS AFRICA

BY

J. H. MIDDLEMISS, M.D., F.F.R., D.M.R.D.
Consultant Radiologist, United Bristol Hospitals

In February 1953 I set out on a three-months trip for the Colonial Office. I visited Sierra Leone, the Gold Coast, Nigeria, Northern Rhodesia, Tanganyika, Zanzibar, Kenya and Uganda; and in addition to these territories under the administration of the Colonial Office, I also visited the Union of South Africa and Southern Rhodesia. The object of the trip was to meet anybody who was practising X-ray work in any form, to discuss their problems with them, and if possible to help them. Such help ranged from showing a medical officer in a bush station how to use his X-ray apparatus to advising a Government what apparatus to buy, and from trying to arrange maintenance services on a zonal basis for Africa to interpreting the films of abstruse diseases. It was useless to think of X-ray work for say, Nigeria or Tanganyika in the same terms and on the same scale as in this country. It was necessary first to see radiology in relation to the type of work carried out in the hospitals and it was necessary to see hospital practice against the background of community life, social conditions, tribal customs, standard of education and so forth. As a result I spent much time in bush villages watching meals being prepared in mud-huts, in shanty towns looking at living conditions, with sanitary inspectors seeing conservancy schemes or malaria control schemes, and with Ministers of Health discussing their plans. And in order to get a true impression of the pattern of disease I spent as much time seeing patients in native dispensaries and hospital " casual " or out-patient departments as I spent going round hospital wards.

The pattern of disease that came within my sphere fell broadly into two categories—trauma and pulmonary tuberculosis. Pulmonary tuberculosis is widely disseminated particularly where life has become at all " urbanized ". At present the African appears to have little resistance to tuberculosis and a progressive primary infection with cavitation is a common response to infection. Often by the time it comes to light the disease has progressed far beyond a stage from which there is any reasonable chance of recovery. At present, the extent of the problem is not even known; a survey is being planned in the Gold Coast to determine how widespread pulmonary tuberculosis is in the territory; similar surveys are under way in various parts of Nigeria and also in Northern Rhodesia. It is hard for us in this country to realize what problems such a survey may produce; first of all there are difficulties in persuading tribal peoples to submit themselves to X-ray examination, and in getting them to co-operate in such a simple procedure as holding their breath; and there is the by-no-means small problem of carrying heavy " mobile " X-ray equipment where no roads or bridges but only dirt tracks and ferries exist. Previously no one radiologist had visited all these territories. But now the experience gained in one territory can be put to use in another, the teething troubles with new apparatus in one place can be avoided in another, and tribal difficulties in one area can be anticipated elsewhere, and so forth.

Trauma, the other major source of X-ray work is found in all territories. In many hospitals " lorry accident " is a diagnosis, and the most appalling examples of fractures are to be seen wherever there is motor traffic. Railways and docks, too, produce their share of fractures. In Sierra Leone and again in Zanzibar, I met what amounted to an occupational disease—fracture of the spine sustained by falling from palm-trees while collecting either palm-kernels or coconuts.

Apart from trauma and pulmonary tuberculosis, X-ray work varies from place to place depending on the tribal customs, local industries and the interests of the local doctors. If, for instance, the surgical specialist in a centre is interested in repairing vesico-vaginal fistulae he will find unlimited clinical material and he will call very little on the X-ray service. If, however, he is interested in orthopaedics, again he will have unlimited clinical material, and in addition he will make heavy demands on the X-ray service. If the local industry is banana growing as in Uganda, or clove production as in Zanzibar, or cocoa growing as in Ashanti province of the Gold Coast, there will be little in the way of industrial disease. But in the copper belt of Northern Rhodesia, where mining is extensive, silicosis is a very real problem. An elaborate Silicosis Bureau has been established at Kitwe with modern X-ray apparatus and a team of experts who work entirely on this problem. In Tanganyika there are gold-mines south of Lake Victoria; so far it is not known whether there is any silicosis there. But a scout survey of some 500 miners is being carried out along lines suggested by me and I shall interpret the films. From this it is hoped to learn whether a silicosis problem exists and whether or not any preventive measures are to be taken. Such a survey, of course, is not the simple procedure that it would be in this country; there is no available electricity supply so a small petrol-engine generator has to be carried; the Africans being examined are suspicious and not always co-operative; X-ray film fogs easily under these conditions and has to be treated carefully; there is no dark-room available and a mud-hut has to be adapted to the occasion; processing of exposed X-ray films should be carried out at a temperature of 65–68° F.—under these conditions it will have to be done at about 85° F.

The recruitment of technical staff from home or the training of Africans to use X-ray apparatus are both subjects which cannot be regarded as simple straightforward problems. I visited some hospitals for instance where the very busy medical officer was trying among his many duties to do the X-ray work. Often he had not been trained for the work nor could he always interpret the results; and usually he had insufficient time to devote to it anyway. The answer in such places would be temporarily to recruit a European radiographer to do the technical work. But a European technician would need a house to live in and this building would be low on the priority list, for in some instances there are not houses even for doctors (in one Mohammedan village I found the doctor living in an " African round-house "). It would take three years to train an African for the work provided a vacancy can be obtained for him in one of their few training schools. Few natives have the right educational standard. In Nigeria, for instance, in a population of 30,000,000 there are 30,000 people with School Certificate; they are the "cream " of the educated people and they are or will become the professional classes, so they are not prepared to remain technicians; those who have not reached School Certificate standard are usually far behind, or have had no schooling at all; they are unable to read or write, and sometimes they cannot even speak English. Obviously it is not possible to teach them the elementary anatomy or apparatus construction to cope with even the simplest aspects of this technical work.

The countries that I visited are developing rapidly and African consciousness is awakening everywhere. But in many ways Africans are still centuries behind us; and they have not yet the education, money or facilities to benefit from all the advances of modern medicine. We have a moral obligation to train these people, to guide them and to let them have the fruits of our experience. In the field of radiology, one of the most expensive in the whole of medical practice, they need much sound advice and guidance so that the plans are well laid and the growing facilities are used to the best advantage.

MONGOLISM, HYPERTROPHIC PYLORIC STENOSIS TREATED WITH "SKOPYL" AND DEATH FROM MILIARY TUBERCULOSIS AND MENINGITIS*

Dr. Beryl Corner: This baby was born in hospital and was recognized as a mongol shortly after birth. His progress during the first fortnight was reasonably satisfactory. When he was three weeks old he started vomiting and was readmitted aged five weeks. On readmission the child was dehydrated and there was clear clinical evidence of pyloric stenosis. Medical treatment was started using the drug "Skopyl", which is prepared in Sweden and has been tried out on a research series of cases of infantile pyloric stenosis in Bristol. This case was included in the research group. The baby was perhaps a little slower to respond than some, and a little vomiting continued for the first few days of treatment, whereas most babies stop vomiting at once. Subsequently all vomiting ceased and he made good progress. The baby was a severe example of mongolism with flabby musculature and poor appetite, but good progress was eventually made and he went out of hospital gaining weight, taking feeds well and in every way, clinically, a healthy baby. Children in this series were seen regularly up to two years after treatment to ensure that the condition had been cured rather than symptomatically relieved; therefore this child was seen at intervals and X-rays demonstrated satisfactory functional progress. I saw him up to June, 1953, when he was about eighteen months old; he was doing quite well despite the fact that the mother was not very intelligent and the baby often appeared looking dirty. There were no symptoms and when he was X-rayed in July, stomach emptying time and the pyloric canal were both apparently normal.

I had made an appointment to see him in November, but he did not keep the appointment and the Health Visitor reported that the baby had bronchitis. He was admitted to hospital as an emergency in a moribund condition at the end of November. The admission diagnosis was acute bronchopneumonia; râles were heard all over his chest; he was in a moribund condition and died shortly afterwards.

Professor Hewer: Would Dr. Corner tell us something about "Skopyl" and its use?

Dr. Corner: "Skopyl" is a trade name for methylscopolamine nitrate. The important point about this drug is that the methylation of the scopolamine makes its peripheral effect on the alimentary tract very much intensified as compared with the central action. This drug was used experimentally in Scandinavia, not originally for pyloric stenosis, but for relief of various types of colic, and was found to be more effective than methyl atropine nitrate in its action on the gut so that it was eventually used for congenital hypertrophic pyloric stenosis in infancy. I have treated 110 cases with this particular drug since 1949, and have not submitted one of these babies to operation since the first three months of the research trial. Clinically, the effect is quite dramatic. The majority of babies stop vomiting within three days and about 35 per cent. never vomit again after the drug has been started. I have seen no toxic effects from it in the dosage which we give, which has been a matter for experimentation. Treatment is given regularly until the child is about sixteen weeks old, reducing the dose gradually towards the end of the period of treatment, since at sixteen weeks, without any treatment, pyloric stenosis symptoms usually begin to lessen. "Skopyl" has proved extremely satisfactory in this clinical trial; most of the cases have been

*A case report at a Clinico-Pathological Conference held on 23rd February, 1954, in the Department of Pathology, University of Bristol.

followed up clinically and radiologically and this is the only one who has died, and therefore it is the only one in which we have had a chance to examine the pyloric canal after treatment.

In reply to a question by Professor Bruce Perry, Dr. Corner said the heart was normal.

Dr. B. E. McConnell: Is "Skopyl" used in the treatment of pertussis, as is eumydrin?

Dr. Corner: Not so far as I know: it is in very short supply.

Dr. G. R. Airth: The initial findings in December 1951 were typical of developed hypertrophic pyloric stenosis. In July 1952, the pyloric canal was still long, narrow and irregular but gastric emptying commenced immediately. In November 1952, the canal was shorter but still of irregular diameter. In July 1953, the canal showed a well-defined mucosal pattern throughout and the stomach emptied rapidly.

These findings indicate successful medical treatment of the spastic obstruction, but it must be realized that they give no information concerning the regression or persistence of the pyloric tumour.

Dr. H. Urich: The post-mortem findings can best be discussed under three headings: (1) mongolism, (2) congenital hypertrophic pyloric stenosis, and (3) the terminal infection.

(1) The body was rather small for a child of two, and weighed 8,400 gm. It showed typical mongoloid stigmata: a small, round skull, eyes set widely apart with epicanthic folds, a small nose with a flattened bridge, short square hands with simian creases (on the left palm only) and short curved little fingers. The heart showed no congenital malformation. I could not detect any gross abnormalities in the brain other than those due to the terminal infection.

(2) The stomach was of normal size and shape. It showed no dilatation or hypertrophy in the body of the stomach, and the mucosal pattern was normal. The pyloric canal was elongated. Its length was 2 cm.; its wall was grossly hypertrophied and 3 mm. thick. The lumen was adequate and the mucosal pattern normal. (Plate XVI.)

Histological examination showed that the elongation and thickening of the wall of the pyloric canal was due entirely to gross hypertrophy of the circular muscle, typical of congenital hypertrophic pyloric stenosis.

It therefore appears that in spite of a complete functional recovery the underlying structural abnormality remained largely unaffected by treatment.

(3) The right lung showed a round, whitish, caseating nodule 1 cm. in diameter, in the upper lobe. The corresponding peribronchial lymph nodes as well as those in the mediastinum were grossly enlarged and showed extensive caseation. The appearances were typical of a primary tuberculous complex. (Plate XVII.)

The remaining parts of the lung showed numerous small whitish or greyish nodules, about 1 mm. in diameter. Similar lesions were found in the liver and kidneys, as well as in the spleen where they were somewhat larger and showed caseation in the centre. The lesions had the appearance of miliary tubercles.

The brain showed some flattening of the convolutions. The base of the brain was covered with thick gelatinous exudate which spread symmetrically around the brain stem, extended into the lateral fissures and over the roof of the fourth ventricle. Minute whitish granules were visible in some parts of the exudate, particularly along the course of the vessels. Cut sections of the brain showed internal hydrocephalus with dilatation of all four ventricles. The appearances were those of tuberculous meningitis.

Histological examination confirmed the tuberculous nature of all the lesions. Sections of lung, liver and spleen showed typical miliary tubercles consisting of lymphocytes, epithelioid cells and giant cells of the Langhans type. Larger foci showed extensive caseation. The meninges showed extensive infiltration with lymphocytes and epithelioid cells, extending into the adventitial spaces around the cortical vessels. The arteries in the affected meninges showed arteritis with

proliferation of the intima and almost complete occlusion of some of the smaller vessels.

Sections stained by Ziehl-Neelsen's method showed scanty acid-fast bacilli in the lesions.

To sum up, the case was one of mongolism with congenital hypertrophic pyloric stenosis, from which he made a good functional recovery, and death from generalized miliary tuberculosis and tuberculous meningitis.

Dr. Corner: Following this post-mortem report a search for contact with an open case of tuberculosis revealed that a friend of the family, who had been visiting them almost daily during August 1953, was subsequently proved to have severe open phthisis from which she had since died.

Dr. R. L. Bishton: Did the brain show any sign of mongolism?

Dr. Urich: As I have not been able to find a clear description of the changes in the brain in mongolism, I would rather not answer that question.

Professor Hewer: Dr. Norman is an expert on the pathology of mongolism: will he kindly tell us about it?

Dr. R. M. Norman: Dr. Urich is right when he says that the text-books give little consistent guidance as to the neuropathology of mongolism. As a matter of fact, the naked-eye appearances of the brain in mongolism are much more characteristic of the condition than are the microscopical. The small round mongol skull owes its characteristic shape to a post-natal retardation of growth of the cartilaginous base and this abnormal shape imposes itself upon the brain. It is unusual to find the brain weight much above 1,100 gm., and in the majority the weight is nearer the thousand mark. Viewed from the side the contour of the occipital lobe rises almost vertically from the pole and the frontal lobes are stunted so that the whole brain looks globular. A common feature is the narrow, poorly developed superior temporal gyrus which is present in more than half the cases. A less common but well established abnormality is a relatively small cerebellum and brain stem—a persistence of the state of affairs normally present at birth and another instance of the "foetalism" of the mongol. Most writers speak of the simple convolutional pattern in this condition. While this impression is usually given by superficial examination, one often sees on section that the larger gyri are mushroomed at the crowns and overlap smaller, hidden convolutions. It seems that there is a real crowding together of the gyri and at times one may even see an actual fibrous fusion at points in the outer layers of contiguous convolutions.

It is commonly stated that the nerve cells are grossly diminished in the mongol cortex and at first sight it seems a reasonable notion that such low-grade defectives should have fewer neurones than the normal. In point of fact it is quite difficult to demonstrate areas where an obvious reduction in nerve cells has occurred—at any rate in mongols who have survived infancy, and it is certainly true to say that such a finding is not a widespread or a constant feature. No doubt circulatory anomalies especially those associated with congenital heart disease may account for the patchy "areas of clearing" sometimes present and perhaps smaller factors underlie the mild fibrillary gliosis often found in the white matter. In general, however, cell counts made in corresponding cortical areas in mongol and normal brains reveal either no difference or even an increase in favour of the mongol. This is due to a patchy clumping together of relatively immature nerve cells. It may well be that the total number of nerve cells in the mongol brain equals the normal but that, corresponding to the small over-all size of the brain, these nerve cells are more crowded together, are less well developed individually and have, therefore, less opportunity for synaptic connection with each other.

One other feature of interest may be mentioned. It is well known that the mongol ages prematurely and this is reflected in the brains of some of those who reach the forties by the presence of senile plaques and the neurofibrillary change of Alzheimer.

Annotation

BUNGALOWS FOR DISABLED PERSONS

The Urban District Council of Burnham-on-Sea has provided a group of bungalows specially designed for disabled persons. The following notes give a brief description of these:

1. They have been primarily designed on the assumption that the disablement necessitates the person using a wheel-chair.
2. There are no steps in the approach to the bungalow, ramps being provided.
3. A verandah outside the entrance gives a convenient place in which to leave the wheel-chair.
4. The entrance hall is very wide, enabling a wheel-chair to use the hall readily and to turn around within its width.
5. All doors are of sufficient width to allow the passage of a wheel-chair.
6. Three bedrooms are provided, but as a disabled person might work at home it is understood one bedroom can be used as a workroom.
7. The water-closet compartment is wide and is provided with chromium hand-grips, one on each side of the pan.
8. The bathroom is provided with fittings which the disabled person can use to help himself into the bath.
9. In one bedroom a hand-grip is provided which the disabled person can use to help himself into bed.

Notes and News

Mr. W. A. Jackman, F.R.C.S. (Ed.), has been elected to the Fellowship of the Royal College of Surgeons of England *ad eundem*.

Dr. J. M. Naish has been elected to the Fellowship of the Royal College of Physicians of London.

Mr. J. H. Peacock, F.R.C.S., has been awarded the Jacksonian Prize of the Royal College of Surgeons of England.

Dr. J. Creighton Bramwell, of Manchester, will deliver the Carey Coombs Memorial Lecture in 1954 on " Rheumatic Valvular Disease 1924–54 ".

Mr. Norman Tanner, F.R.C.S., will deliver the Long Fox Memorial Lecture on a subject to be announced later.

Professor Bruce T. Mayes, Professor of Obstetrics in Sydney, gave a lecture at Southmead on May 16th on " Post-partum Haemorrhage and the Management of the Third Stage of Labour ".

A week-end course for general practitioners was held at Exeter in the week-end May 14th–17th. A week's course will be held in Bristol from July 11th to 17th. Next session the usual series of Sunday morning demonstrations will probably be arranged in Bristol.

Messrs. John Wright & Sons have inaugurated a travelling book showroom for the sale of medical books to the staffs of peripheral hospitals in the South West.

EXAMINATION RESULTS

M.R.C.P. (London): M. D. Turner.
F.R.C.S. (Ed.): E. R. Duchesne.
D.A.: C. D. Linton.
D.P.M. (Bristol): L. W. Bowen, D. E. Jarman, S. Prus.
D.C.H.; Miss A. M. Haines, Miss H. Patel, Miss F. E. Powell, Mrs. R. M. Walters, G. R. Wijegunaratne.

PRIZES

Francis Dudley Memorial Prize for 1953: Awarded jointly to Miss D. O'Connor and Mr. M. G. Addy.
Dental Gold Medal: Awarded to Mr. J. E. Bowerman.
Hewer Pathology Prize for 1954: Awarded to Mr. G. A. J. Ayliffe and Mr. K. R. Gough.
Foyle Prize for 1954: Awarded to Mr. R. T. Marcus.

Appointments

UNITED BRISTOL HOSPITALS

Name	*Specialty*	*Where from*	*Commenced*
MACGILLIVRAY, IAN, M.B., CH.B., M.R.C.O.G., M.D. (*Hon. Appointment*)	Obstetrics and Gynaecology.	Was previously lecturer in Dept. of Midwifery, University of Glasgow, and Clinical Assistant, Glasgow Royal Maternity and Women's Hospital and Western Infirmary, Glasgow.	1.1.54
PRICE, ARTHUR KENNETH, M.B., B.S., F.R.C.S., M.R.C.S., L.R.C.P. (*Hon. Appointment*)	Thoracic Surgery.	Consultant in Thoracic Surgery, South Western Regional Hospital Board (Frenchay Hospital).	1.1.54

Name	*Previous Post*	*Post appointed to*	*Commencing date*
CAMPBELL, MISS D. M. Y., L.D.S.	Senior Orthodontic Registrar, King's College Hospital Dental School.	Senior Registrar in Orthodontics.	Jan., 1954
LEATHER, H. M., M.B., CH.B., M.R.C.P.	Medical Registrar, Manchester Royal Infirmary, and Resident Medical Officer, Barnes Hospital, Cheadle.	Senior Medical Registrar.	Mar., 1954
HILL, R., M.B., CH.B., M.R.C.P.	Medical S.H.O., Southmead Hospital, Bristol.	Medical Registrar.	Mar., 1954
JOHNSON, D. H., M.B., CH.B., M.D.	Registrar in Pathology, United Bristol Hospitals.	Senior Registrar in Pathology.	Mar., 1954
MOORE, G. E., L.D.S., H.D.D., F.D.S.	Dental House Surgeon, Worcestershire, Mid-District.	Registrar in Dental Surgery.	Mar., 1954
MONKS, P. J. W., M.B., B.S., F.R.C.S.	Surgical Registrar, Southmead Hospital, Bristol.	Senior Surgical Registrar.	Apl., 1954
CROW, R. S., M.B., CH.B., M.R.C.P.	Senior Medical Registrar, Kingston Hospital, Surrey.	Senior Medical Registrar.	Apl., 1954
FORREST, MISS G. M. S., M.B., CH.B.	Senior House Officer Anaesthetist, Cheltenham General, Eye and Children's Hospital.	Registrar in Anaesthetics.	Apl., 1954
NOBLE, D. S., M.B., B.S., B.D.S., L.D.S., R.C.S.	House Surgeon, Guy's Hospital Dental School.	Registrar in Dental Surgery.	Apl., 1954
SPENCER, R. W., M.B., CH.B., M.R.C.P.	Junior Assistant Pathologist, John Bonnett Laboratory, Cambridge University.	Senior Registrar in Pathology.	May, 1954
MOORE, E. W., M.B., CH.B., D.P.H., M.R.C.P.	Part-time Registrar in Diagnostic Radiology, United Bristol Hospitals.	Registrar in Radiology (Diagnostic).	June, 1954
HARDY, H. M., M.B., CH.B.	Registrar in Anaesthetics, United Sheffield Hospitals.	Registrar in Anaesthetics.	June, 1954

BRISTOL

Name	*Appointment*	*Formerly*
LITTLE, BRIAN R., M.R.C.S., L.R.C.P., D.A.(ENG.), F.F.A.R.C.S.	Consultant Anaesthetist, Bristol Clinical Area (Weston-super-Mare).	Consultant Anaesthetist, North Gloucestershire, Clinical Area.
SLEIGH, BEATRICE E., M.B., CH.B., D.A.	Anaesthetic Registrar, Frenchay Hospital, Bristol.	Anaesthetic Registrar, United Bristol Hospitals.
TULLOCH, L. G., M.B., CH.B. (ABERDEEN), M.D. (ABERDEEN).	Senior Registrar in Dermatology (Joint appointment with the United Bristol Hospitals).	Registrar in Dermatology, Manchester and Salford Hospital for Diseases of the Skin.
HOUGHTON, E. A. W., M.B., CH.B. (BRISTOL).	Medical Registrar, Southmead Hospital, Bristol.	S.H.O. in General Medicine, Southmead Hospital.
KNOWLES, R. R., M.B., B.S. (SYDNEY), D.P.M.	Senior Registrar in Psychiatry, Bristol Mental Hospitals.	Psychiatric Registrar, Blackburn Royal Infirmary.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
REES, DAIREEN I., M.R.C.S., L.R.C.P.	General Practitioner Anaesthetist, Stroud General Hospital (2 sessions).	Is in general practice at Nailsworth.
ROWLANDS, IRENE P., M.R.C.S., L.R.C.P., F.R.F.P.S., M.R.C.P. (EDIN. & LONDON).	S.H.M.O. Geriatrician, North Gloucestershire Clinical Area.	Medical Registrar, Hereford Group of Hospitals.

BATH

BURROWES, W. L. M.B., B.CH. (BELFAST), M.D., M.R.C.P.I.	Clinical Assistant in General Medicine, Royal United Hospital, Bath (1 session).	Is in general practice at Corsham.

EXETER

Name	*Appointment*	*Formerly*
TROBRIDGE, G. F., M.B., CH.B. (B'HAM).	Registrar in Diseases of the Chest, Hawkmoor Chest Hospital, Bovey Tracey.	Registrar, Leeds Chest Clinic.
DORAN, H. J., M.B., B.CH. (BELFAST), D.L.O.	Clinical Asst. in E.N.T. Surgery, Newton Abbot and Dawlish Hospitals (2 sessions).	Is in general practice at Torquay.

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
BRAMLEY, P. A., L.D.S., R.C.S. (ENG.), L.D.S. (B'HAM), M.B., CH.B. (B'HAM), F.D.S., R.C.S. (ENG.).	Consultant Dental Surgeon, Plymouth Clinical Area (4 sessions).	Dental Registrar, Rooksdown House, Plastic and Jaw Unit, Basingstoke.
COLLINS, JOAN R., M.B., CH.B. (B'HAM).	General Practitioner Anaesthetist, Newquay and District Hospital (2 sessions).	Is in general practice at Newquay.
TRUSCOTT, D. E., M.B., B.S., D.M.R.(D.) (LOND.), D.M.R.(D.) (CONJOINT BOARD).	Consultant Radiologist, based at West Cornwall Hospital, Penzance.	Senior Registrar in Radiology, Glasgow Royal Infirmary.

Book Review

"ARTHUR RENDLE SHORT", by W. M. CAPPER and D. JOHNSON. Published by the Inter-Varsity Fellowship at 8s. 6d.

Many old students of Bristol and others who knew Rendle Short will wish to possess this book. The writing of it was obviously a labour of love for the authors and they have admirably discharged their debt to a great Christian teacher of Medicine. In addition they have retold many excellent stories which he used to give point and interest to his teaching.

DAMNED DRUGS

PART II

INAUGURAL PRESIDENTIAL ADDRESS

Given at The Royal Fort, Bristol, on 8th October, 1952

BY

DR. G. E. F. SUTTON, M.C., M.D., F.R.C.P.

Chloroform and the Pimlico Mystery

It is rare to find an ether addict, but occasionally one meets with an individual who is addicted to chloroform. I knew of a doctor who put himself to sleep every night by placing a handkerchief saturated in chloroform over his face. This is a very dangerous thing to do and it proved eventually to be fatal in his case. In the so-called Pimlico mystery it was claimed that chloroform was used with homicidal intent.

In 1875, Thomas Edwin Bartlett married a young Frenchwoman, Adelaine Blanche de la Tremoille, aged twenty, who was a native of Orleans. After their marriage Bartlett arranged for his wife to go to a boarding-school, so she lived with him only during the vacation. He then sent her to a convent school in Belgium, after which she settled down with her husband in London where he had several grocer's shops. Six years after their marriage Mrs. Bartlett gave birth to a stillborn child, and she was so upset by this event that she decided she would never have any more children. Four years later she and her husband made the acquaintance of a young Wesleyan minister, George Dyson, for whom they both conceived a great admiration. It was not long before they were on terms of what has been described as great social intimacy, and in 1885 Edwin Bartlett made a will leaving all he possessed to his wife and naming Mr. Dyson and his solicitor as his executors.

On December 10th of the same year Mr. Bartlett became seriously ill, but the astute practitioner who was called in to attend him thought that the state of the gums and the peculiar nature of the symptoms pointed to poisoning with mercury. He seemed to be gradually recovering, but nine days later he asked that a second doctor should be called in, " lest ", as he said, " if anything happened to me my friends should suspect that my wife was poisoning me ". Later it was said in court that this action was the result of some ill feeling which had formerly existed between his wife and his father.

The second practitioner was duly called in, and on December 26th, though still feeling weak, Mr. Bartlett had so far recovered as to be able to go for a drive.

On the following day Mrs. Bartlett asked Mr. Dyson (during one of his constant visits to the house) to procure for her a considerable quantity of chloroform. She told him, so the story goes, that her husband was afflicted with an internal complaint which at times caused paroxysms. She also expressed the opinion that he might die suddenly in one of these attacks. Mr. Dyson obtained three lots of chloroform—in all six ounces—from various chemists, stating that he required it for removing grease spots! Two days later he met Mrs. Bartlett on the embankment and handed it to her.

During his illness Mr. Bartlett slept on a camp-bed in the drawing-room and his wife slept on the sofa there. On December 31st he was quite fit again and at half-past ten on that evening Mrs. Bartlett told the maid that she required nothing else and retired with her husband for the night. At 4 in the morning she aroused the house,

and it was found that her husband was dead in bed. She said that, after she had settled her husband for the night, she sat at the foot of the bed with her hand resting on his feet. After a time she dozed off and later awoke with a feeling of cramp and was horrified to find that her husband's feet were deathly cold. She tried to pour some brandy down his throat but found he was dead. She then aroused the household. The first person to enter the room was the landlord, who noticed a peculiar smell like " chloric ether ".

The doctor was sent for at once, but could find nothing to account for death. A search was made, but the only bottle found was one that contained a few drops of chlorodyne. Autopsy showed that all the organs were healthy, but the stomach showed evidence of having contained a lot of chloroform.

The coroner's inquiry resulted in a verdict of wilful murder against Adelaide Bartlett and George Dyson and they were both arrested. But this was not the end. At the trial the Crown decided to offer no evidence against Dyson and he was duly called as a witness. Sir Edward Clark, who was said to be able to reduce any jury to tears, was called for the defence of Mrs. Bartlett.

The Rev. Mr. Dyson's examination occupied nearly the whole of the second day and he disclosed in some detail the form of intimacy between Mrs. Bartlett and himself. He admitted that he had purchased the chloroform and after hearing the result of the autopsy, had thrown the bottles away on Wandsworth Common while on his way to preach at Tooting. He said he had been in the habit of kissing Mrs. Bartlett and that her husband thoroughly approved of it. He had told him that he was becoming attached to his wife, but Mr. Bartlett had actually encouraged him and told him to continue the intimacy. The Rev. Mr. Dyson admitted that he did not tell Mr. Bartlett that he had procured the chloroform.

The doctor who had attended Mr. Bartlett then gave an account of an extraordinary statement which was made to him by Mrs. Bartlett after the death of her husband.

She said that she had been selected by Mr. Bartlett at the age of sixteen as a wife for companionship only. There was a sort of marriage compact in which they were to live together as loving friends and which precluded any carnal feeling. This strange and far from natural alliance was only terminated at the end of six years at Mrs. Bartlett's earnest and repeated entreaty that she should be allowed to become a wife and mother. The result of this late union was a stillborn child, and from that time onward there was no intercourse. Mrs. Bartlett further averred that her husband lavished on her an ultraplatonic affection. He encouraged her to pursue various studies, and liked to surround her with male admirers. She maintained that he encouraged the Rev. Mr. Dyson and herself to kiss in his presence, and not only did he enjoy it, but he gave her to understand that he had given her to him. Then, as her husband recovered from his illness, he expressed the wish that they should resume the ordinary relationship of man and wife, but we are not surprised to learn that she resented it and that she accordingly sought some means of frustrating his desire. It was thus that she asked her paramour to procure the chloroform.

On the night of her husband's death there had been some conversation of this sort, and when he was in bed she brought the bottle of chloroform and gave it to him, telling him that she intended to sprinkle some on his handkerchief and wave it in front of his face so that he might go peacefully to sleep. He looked at the bottle and placed it by the side of the camp-bed on which he was lying and, turning over on his side, apparently went to sleep. She also went to sleep sitting at the foot of the bed with her arm around his foot. Later, she heard him snoring, then woke again and found he was dead.

There can be little doubt that the recounting by the medical attendant of this strange history must have played no small part in the ultimate finding of the court. Certainly it enabled Sir Edward Marshall Hall to give full rein to his wonderful histrionic powers, and it is not difficult to visualize his listening to its unfolding with a relish that even this great dramatic criminal lawyer can rarely have known.

Dr. Stevenson stated that he found eleven and a quarter grains of pure chloroform

in the stomach of the deceased, but judging by the time that had elapsed and the very volatile nature of the fluid, a large quantity must have been swallowed. He found no other poisons.

After deliberating nearly two hours the jury returned a verdict of " Not Guilty "!

Since the Pimlico poison case must remain among the unsolved mysteries, it is interesting to speculate on a few salient features which do not appear to have been sufficiently stressed.

It is well to recall, for instance, that when he was first called in to attend Mr. Bartlett on December 10th, the practitioner thought the state of his gums, together with the other symptoms, pointed to poisoning with mercury. Then why did the patient suddenly ask for another practitioner and give as his excuse for doing so: " If anything should happen to me my friends might suspect that my wife had been poisoning me "? Again, for what purpose did Mrs. Bartlett require chloroform? Why did she say that her husband had an internal complaint which caused paroxysms which she believed might end fatally? Yet Mr. Bartlett had been healthy in the past and the autopsy showed no evidence of internal disease. Again, in one breath we are told by a thinly veiled statement that there had probably been no marital relationship since the birth of her stillborn child, and that Mr. Bartlett's attitude towards his wife was so ultraplatonic as to be quite quixotic; yet, in the next breath, we are given to understand that, after his recovery, he wanted to resume normal marital life, much to the dismay of his wife.

Turning now to the Rev. Mr. Dyson, why did he buy chloroform from various chemists—six ounces of it—and why did he tell them that he wanted it for removing grease spots? (It is possible that even Mr. Bartlett might have taken some exception to being so described!) Why, when he heard of Mr. Bartlett's death, did he consider it necessary to dispose of the bottles on Wandsworth Common? We might also inquire why he purchased three lots of chloroform from three different chemists and then poured it all into one bottle.

We are informed of the peculiar attitude of Mr. Bartlett, of how he encouraged the intimacy of his wife and the Rev. Mr. Dyson, even approving of their kissing in his presence. Even assuming that he was somewhat quixotic, however, it is well to reflect that, had he been alive to do so, he might have thrown a very different and possibly more revealing light on the scene. He might, for instance, have smiled benignly at a friendly peck between his wife and the reverend gentleman who was his trusted friend. But even lovers are apt to embrace rather shyly in the presence of another, especially if that person happens to be the husband.

Be that as it may, we can, nevertheless, find some solace in the thought that had Mr. Bartlett died under similar circumstances to-day, an up-to-date counsel for the defence would have painted him with lurid colours as such a repulsive, perverted monster that the disgusted jury would have felt impelled to bring in a verdict of " Not Guilty ".

We are not told whether Mrs. Bartlett and Mr. Dyson lived happily ever after, but we do know that she inherited all her husband's considerable possessions.

Some of you might think that the Scottish verdict of " Not Proven " would have been at least more appropriate than " Not Guiity " in this somewhat tangled case.

Deep-dyed Death from Diphtheria

Both typhoid and cholera germs have been used for homicidal poisoning, but one of the most ingenious crimes on record was that in which diphtheria toxin was employed as the death-dealing agent.

In Petrograd in 1911 there lived a ne'er-do-well, Patrick O'Brien de Lacy, who claimed to be a lineal descendant of the Irish kings. Having met a lady of excellent family he easily persuaded her to divorce her husband and marry him. He subsequently spent all her money and then divorced her. Finally, having made the acquaintance of a Mlle Buturlin, he sought out a doctor, Panchenko, and conspired with him to poison the lady even before they were married.

Panchenko was a needy practitioner and O'Brien de Lacy offered him 10,000 roubles for the death of his brother-in-law, 50,000 roubles if he subsequently disposed of his father-in-law, and 500,000 roubles if he then dispatched his mother-in-law who was the wealthiest of the family.

In 1910 the younger Buturlin died at Petrograd after a week's illness. His father, old General Buturlin, who arrived from Vilna before the funeral, stopped the interment and demanded an autopsy. This being done it was decided that death was due to blood-poisoning and no further inquiry would have been made but for the fact that on that same day a man named Bobroff, who was employed by Panchenko as a tout to bring him patients, made a statement to the Chief of the Secret Police:

" A conscience is the only possession I can call my own, and it has driven me here to denounce my unique benefactress. She is my landlady, Mme Muraviova, who allows me a room in her flat and has been very kind to me. She is the mistress of Dr. Panchenko, with whom she has been hugger-muggering of late in suspicious ways. The door and walls being thin, I have heard snatches of conversation, which I have pieced together and I find that they point to Dr. Panchenko as the instrument of young Buturlin's death, and to O'Brien de Lacy as the employer of that instrument. The penniless Dr. Panchenko often journeyed to Vilna where O'Brien de Lacy resides and always returned with a fat purse and high hopes. Mme Muraviova, too, babbled about her improving prospects, saying she was shortly coming into 300,000 roubles. One day in April, Dr. Panchenko left for Kronstadt, where plague-stricken dogs are studied, and after his return he talked of little else. Soon afterwards young Buturlin, Panchenko and O'Brien de Lacy went on the spree together. The next thing I noticed was that Panchenko was weeping and sobbing. I entered the sitting-room and found him beside himself with excitement while his paramour was burning heaps of papers. She spoke first, saying to me that she had been scolding him for visiting a diphtheria patient without disinfecting himself. Then in an aside to Dr. Panchenko she asked: 'Did you do it properly?' He answered: 'Well, I squirted two full doses although one would have been enough'."

As a result of this story both Panchenko and O'Brien de Lacy were arrested. The former, after telling plausible stories, broke down and confessed the whole premeditated crime as follows:

" On May 16th I visited Buturlin and injected pure drug from a phial. I repeated the injection on the following day. Before visiting him in the evening of that day I broke the necks off the two drug phials in my lodgings so that nobody should notice it. Having emptied the contents I filled the phials with diphtheria poison by means of a paper funnel, plugged them with wadding and, putting them in my waistcoat pocket, set out for Buturlin's. Before starting I gulped down vodka for courage. I got to Buturlin's about 8 or 9 in the evening with trembling in my legs and throbbing waves of darkness filling my eyes and fitfully blotting out my sight. I had been wont to break off the phials in Buturlin's presence, first putting them in a handkerchief to avoid cutting my fingers. That is why he could not notice that this time the necks were already snapped off. I made two incisions in Buturlin's body, injecting each time the contents of one phial of the diphtheria poison. As soon as I had finished the business my face was ghastly and I quivered in every limb. I was in dread that Buturlin might discern my state. Pulling myself together and mastering my failing voice, I asked him whether it hurt. He answered: 'Not at all.' I then left for home and threw the phials in the street. The livelong night I could not close an eye. Conscience-ache racked me ruthlessly."

Despite this graphic story, it was subsequently revealed that Panchenko was a hardened criminal who, in addition to many other misdemeanours, had previously planned to poison several other people for money.

As a result of the trial, which lasted nearly three weeks, Panchenko was sentenced to fifteen years' penal servitude, and O'Brien de Lacy was sentenced to penal servitude for life. Panchenko's mistress, Muraviova, was acquitted.

It is interesting to recall that when epistaxis, sharp pains and vomiting ushered in

the fatal agony, young Buturlin exclaimed: "Three months long they were at me to have the injections, but I refused, as though I had a presentiment of what was coming!"

The Smethurst Case

The trial of Dr. Smethurst in 1859 for the murder of Miss Isabella Banks by poison was described by the Lord Chief Justice Pollock, who presided, as one of the most remarkable cases in his then long experience. Dr. Smethurst was at this time fifty-four years of age, though he gave his age as forty-eight. He was a man of small stature and insignificant mien, with a reddish-brown moustache. At the time he met Miss Banks he and his wife (who was twenty years older than himself) were living with her at a boarding-house in Bayswater, London. Their acquaintance ripened so quickly into a warm friendship that the landlady gave Miss Banks notice to quit. Soon afterwards Dr. Smethurst left his wife and joined her. A bigamous marriage took place at Battersea Church and the couple settled down at Richmond.

Miss Banks, aged forty-two, was a woman of some charm and considerable possessions. Apart from occasional bilious attacks she had always been healthy. Soon after her marriage she was taken ill and was attended by Dr. Smethurst who gave her all her food and medicines, and dealt with all the slops. She was complaining of soreness in the mouth and a burning sensation in the throat and intestines, with severe diarrhoea and vomiting, and as she was worse after some days it was decided to call in Dr. Julius, a well-known Richmond practitioner. Before long he began to suspect irritant poisoning and accordingly asked his partner, Dr. Bird, to see her. He, too, came to the same conclusion, but he did not say so until Dr. Julius had divulged his fears to him. During this time Dr. Smethurst continued in his apparently devoted ministrations to his new wife, and he was at some pains to keep her sister away, stating in his letters to her that their dear Bella was really too ill to see her, and as this was the doctor's word, she seems to have accepted it, after the manner of *some* relatives, more or less philosophically.

As Miss Banks's condition steadily deteriorated, Dr. Julius decided to call in Dr. Todd of King's College Hospital, who was one of the most eminent physicians of the day. (The temporary palsy which occasionally ensues after many epileptic fits, you will remember, was described by him and is hence known as Todd's Palsy.) Dr. Todd came to the same conclusion and had the excreta examined by the most distinguished toxicologist of the day, Dr. Alfred Swayne Taylor, Professor of Chemistry at Guy's Hospital. In one of the specimens he found arsenic, so Dr. Smethurst was promptly arrested, but was at once released by the magistrates. Miss Banks died next day and Smethurst was again arrested, this time on a charge of murder.

Two days before her death Dr. Smethurst visited a solicitor in Richmond, taking with him a draft will in his own handwriting, and requested the lawyer to come on the following day (which happened to be a Sunday) to execute it. This he did and Miss Banks, described in the document as a spinster, left everything she possessed —with the exception of a brooch—"to my sincere and beloved friend, Thomas Smethurst".

Autopsy revealed that she was from five to seven weeks pregnant. In the stomach there was a large patch of effused blood, but no signs of ulceration or acute inflammation. There was some inflammation at the commencement of the duodenum, the other parts of the intestine were slightly injected. In the lower three feet of the ileum, the mucous membrane was much thickened. That of the caecum was nearly destroyed by inflammation and ulceration. These appearances decreased along the colon and rectum. No arsenic whatever was found in the body, but antimony, in small quantities, was discovered in the caecum, the small intestine, one of the kidneys—the other not being examined—and in some blood taken from the heart.

The trial which took place at the Old Bailey was interrupted by the sudden severe illness of one of the jurymen and resumed a month later. At the onset there was a

dramatic incident when Dr. Alfred Swayne Taylor admitted that he had made a mistake in saying that arsenic was present in one of the specimens of excreta. He stated that some arsenic was present as an impurity in the copper gauze which he had been using and this had been set free by some potassium chlorate which was present in the bottle in which the specimen was placed.

This resulted in the following letter being written to *The Times* by William Herapath, F.C.S., Professor of Chemistry at Bristol, and grandfather of the late Dr. C. E. K. Herapath, which is so justly scathing and so unanswerable that it must have had no small influence at the time:

" 25th August, 1859.

" I consider that professional witnesses who give their opinions where the life or freedom of a man is at stake are as much upon their trial as the prisoner, and that it is the duty of those who are well acquainted with the particular science they profess to correct any error that might deceive the jury or influence the fate of the prisoner. With these views, from my experience in cases of poisoning, I think I ought to remark upon the chemical evidence adduced in the case of Dr. Smethurst. I find in *The Times* of 21st May that Dr. Taylor deposed that in Bottle No. 21 he found it half-full of a clear fluid, that he tried Reinsch's process on it, and found that there were seven grains of chlorate of potash in an ounce of it, which was 1·6 per cent, and that there was a grain of arsenic to every ounce; he further said that it was of the sort we call white arsenic, and that he had previously tried the tests (materials) and found them pure. Here was evidence of the most positive and distinct kind; not only was arsenic found, but the weight estimated and its nature (white) ascertained. Upon the trial this was stated to be a mistake; that, although the former evidence was given so boldly and so particularly, it was now found that there was no arsenic in it, but that the arsenic found was in the copper, although the copper had, with the other materials, been previously proved to be pure. The witness went on to say that he had at first operated upon an evacuation, and found arsenic in that, but admitted that he had used the same copper for many years; consequently all proof of the presence of the poison either in the body or in the evacuation, or in the bottle in the possession of the prisoner, was destroyed by the witness himself, and the jury must rest upon the symptoms and physiological appearances in the opinions of the medical witnesses called, which are about equal for and against the prisoner. But the mischief does not end here, for if the same impure copper ' has been used for twenty years ', and evidence given upon it, what shall be said of the justice of the convictions and executions which have taken place during those years upon Dr. Taylor's evidence?

" But was the arsenic said to be found in Bottle No. 21 really in the copper used to prove its presence? Could the copper wire gauze dissolved by seven grains of chlorate of potash and its associated hydrochloric acid deposit one grain of arsenic? In the face of all England I say it could not; the hundredth part of a grain of arsenic in that quantity of copper would render it so brittle that it could not be drawn into wire at all, much less into fine wire fit for gauze; the fact is, the whole set of operations were a bungle. Reinsch's process is not applicable where nitrates or chlorates are present.

" Next, where his admirable process is resorted to, the mere discoloration of the copper does not prove the presence of arsenic; it only proves that one or more of the inferior metals—arsenic, antimony, tin, lead, bismuth, mercury, etc., are present.

" To individualize arsenic among these four more experiments are necessary. The first is sublimation in a stream of air, when crystalline arsenious acid is produced from the black deposit. Next, that sublimate must be dissolved in water and tested by three methods: first, ammonia, sulphate of copper; next, ammonia, nitrate of silver; and, thirdly, sulphuretted hydrogen.

" Thus, one separation and four proofs produce a body of evidence which it is impossible to gainsay, and these five proofs should be brought into Court so as to be examined by those who are competent to recognize them; and I here warn juries that no evidence short of tangible production of the poison and its tests ought to be for one

moment attended to; any deviation from this rule will convert a convicted criminal into a martyr, and deprive trial by jury of the infallibility which it ought to possess; and, lest it might be imagined that it may not be possible to secure enough of the poison to make the five proofs, I should say that the one-thousandth part of a grain is quite sufficient."

Then began a conflict of medical evidence the like of which has perhaps never been equalled even to this day. On the one hand there were a number of physicians, including the two practitioners and Dr. Tod, who were convinced that the picture was that of an irritant poison, and on the other hand there were a number of eminent physicians who were equally certain that the symptoms and the autopsy findings were typical of dysentery. Finally, the gynaecologists were quite sure that the patient's symptoms had all been caused by the toxaemia of pregnancy, and they quoted what appeared to be several similar cases in support of their opinion.

Mr. Sargent Parry for the Defence promptly asked Dr. Julius:

" Are you a doctor of medicine? "

"Yes."

" Is yours a London degree? "

" Yes."

" What degree is it? "

" It is the Archbishop of Canterbury's degree."

" What! Can he make a doctor of medicine? "

The Lord Chief Justice: " Yes, and he can also make a Master of Arts."

Mr. Sargent Parry: " Did you take your degree as a matter of course? "

Dr. Julius: " Oh dear, no! It is a very uncommon thing. I had to get a certificate from two members of the College of Physicians stating that they had known me for a length of time and that I was a proper person to have the degree."

Mr. Sargent Parry: " And having that certificate you got the degree?"

" Yes, but it only entitles me to call myself doctor."

The Lord Chief Justice: " But you are a member of the College of Surgeons and a member of the Society of Apothecaries? "

" Yes."

The power to grant medical degrees was a remnant of papal authority which was reserved to the Archbishop of Canterbury by statute passed in 1533 in the reign of Henry VIII. The Medical Act of 1857 abolished any qualification to practise with these degrees unless they were granted prior to the passing of the Act. The Archbishop does not now use his power to confer these empty honours.

In his charge to the jury the Lord Chief Justice left little doubt that the evidence pointed strongly towards Dr. Smethurst's guilt. Apart from the almost immediate illness of Miss Banks following the bigamous marriage, he pointed out that, while Smethurst had kept her sister from the bed of her nearest relative, giving as an excuse the critical state of her health, he yet insisted on the attorney coming in at that moment and against his wishes on a Sunday, to legalize the will which he himself had drawn up in his own handwriting. In that will Isabella Banks was described as a spinster and she appears to have made some feeble protest at this and then signed the will.

The Lord Chief Justice also pointed out the manner in which Smethurst had secluded Miss Banks from everyone, and that on one of the rare occasions when the sister was allowed to see her, she brought some soup which he would not allow to be given, and when she wanted to make her some arrowroot he would not allow her to do so. Again, whereas Miss Banks was vomiting after everything that Smethurst gave her, when Dr. Bird gave her some food and brandy after Smethurst had been arrested the first time, she kept it down. Further, not only was no nurse employed throughout, even for the offices of the sickroom, but when, after his discharge from prison, it was suggested he should employ one, Smethurst refused, saying: " Let Dr. Julius provide these things at his own expense. I am told to interfere no more." Yet he already had £71 of Miss Banks's money in his possession at that time.

It was further pointed out that at the time of his arrest there was found in Smethurst's pocket a letter to his seventy-four-year-old wife, as follows:

" My dearest Mary,
I have not been able to leave for town as I expected in consequence of my medical aid being required in a case of illness. I shall, however, see you as soon as possible, and should any unforseen event prevent my leaving for town before the 11th [the 11th was the day of the month on which payment from Miss Banks's invested money was procured] I will send you a cheque for Smith's money and extras. I will send you £5. I am quite well and sincerely hope you are the same and that I shall find you so when I see you which I trust will not be long first. Present my kind regards to the Smiths and all friends in the house. I heard from James the other day; he said he had called upon you but you had gone out for a walk."

The Lord Chief Justice commented on this to the jury as follows:
" You are to judge whether this is the letter written by a man to his wife whom he has abandoned for ever, or whether it is written by a man who means to go back to her and thinks that the business on which he left her is drawing to a close."
The Lord Chief Justice stated that it may be that no poison is traced to the possession of the accused, and it may be that none is found in the body, and yet it may be the easiest thing in the world to put a case where no sensible man could doubt that the accused had possessed the poison, had used it, and that the deceased had died of it. He added that this is no doubt a case of circumstantial evidence, and it creates a duty on the part of the jury to consider first—" Do you believe the evidence? Do you believe this fact, that fact? The next? And so on; if you do, what do they prove? I can understand it being said: would you find the man guilty for the first? No, certainly not. Would you for the second? No. Would you for the third? No. Or the fourth? No. But what would you do if they were all together? It is really like this—there is a scale with a hundred-pound weight in one of the scales. Will five pounds lift it? No. Will eighty? No. But what will they do altogether? Why, outweigh it abundantly, lift it without any difficulty, and that is really, I think, a just explanation of what may be called the theory of circumstantial evidence. You estimate the value of a fact and it is perfectly indifferent—but coupled with another fact it may be exceedingly important.
The jury returned a verdict of " Guilty ", but, nothing daunted, when called up for judgment, Smethurst launched upon a long harangue on why he was innocent, during which he defamed Dr. Julius in every way possible. Then, when the Lord Chief Justice pronounced sentence of death, he exclaimed: " I declare Dr. Julius to be my murderer. I am innocent before God! "
If the trial had been eventful and even startling at times, it was but a wave in a pond compared with the storm which it evoked. You have already heard William Herapath's scathing denunciation, but both in the lay and medical press there were many more equally scathing, both with regard to the trial and the conviction.
The Secretary of State was petitioned for pardon in a memorial signed by thirty practitioners of London and in yet another signed by twenty-eight members of the bar. Smethurst himself was most active, not only in writing to the *Lancet* and *The Times*, but in petitioning the Secretary of State. His real wife also petitioned on his behalf and she averred that, apart from his one lapse, he had been throughout a loving and devoted husband. His brother, who was a chemist, also wrote to *The Times* on his behalf. Eventually the Secretary of State became so worried with it all that he took the unprecedented action of submitting all the case-notes to the leading surgeon of the day, Sir Benjamin Brodie. In his reply to the Home Secretary, Sir Benjamin made it clear that although the facts were full of suspicion against Smethurst, there was not absolute and complete evidence of his guilt.
The Home Secretary, therefore, advised Her Majesty to grant a free pardon, stating that the necessity of doing so in this case arose from the imperfection of medical science and from fallibility of judgment, in an obscure malady, even of skilful and

experienced medical practitioners. This statement, which was as true then as now, created great offence among the medical profession of the day.

After his release Smethurst was promptly arrested, tried and convicted for bigamy. He was sentenced to one year's hard labour. During this trial it was disclosed that his first wife had committed bigamy when she married him.

Smethurst was no sooner released from prison when he took immediate steps to prove the will of Isabella Banks in his favour. Needless to say, many scathing comments were made by counsel at this hearing, but they fell off Smethurst like water off a duck's back, and in the end he proved the will and retired with the very comfortable income.

In his letter to the Home Secretary, Sir Benjamin Brodie made this comment:

" There is another circumstance which, trivial as it would appear to be, would, in some degree, have influenced my opinion if I had been on the jury, namely, the proposal which Smethurst made to test the strength of the prussic acid which was to be administered to the patient by dipping bread in it to be given to the sparrows and which seems to show that the subject of poisoning was not altogether new to him, and that he must have been in the habit of dabbling in experiments with them."

While I have been engrossed in my grim discourse, many of you, as thoughtful men and women, will have asked yourself the question: " What is the underlying mental make-up of the poisoner, and how does it differ from that of the normal individual? " In his foreword to the book by L. A. Parry, M.D., F.R.C.S., on the Trial of Dr. Smethurst, Mr. John Flowers, K.C., writes these words:

" It has been truly said that most of the interest and part of the terror of great crime are due, not to what is abnormal, but to what is normal in it. Revenge, avarice and passion are often dominating influences in the life of normal persons, and yet one or other of such influences is usually the motive for murder. The normal person in this world has, therefore, something, however slight, in common with the criminal, and naturally looks with interest and amazement at the tragic depths to which a man may sometimes fall under the influence of motives which are common to his fellow men. If this is not so, it is a little difficult to understand the extraordinary outbursts of feeling in favour of a person who is suspected of having committed a murder. For this reason he sometimes becomes a popular hero, quite apart from the merits or demerits of the case which is made against him, and his acquital, which may often mean no more than a case of not proven, is made the occasion of misplaced and ridiculous hero worship."

We might well add that " since all the world's queer except thee and me, and even thee's a bit queer ", " there, but for the Grace of God, goes John Bunyan ", preceded by all and sundry. But the problem of the psyche and its motivation is not a simple one. The late Justice Darling, when asked the difference between the mind of the murderer and that of the normal individual, stated that the murderer was a sublime egoist, so that everything had to be sacrificed to the attainment of his selfish ends. The learned judge did not consider that he differed materially in other ways from normal individuals. Certainly there is much egocentricity in normal people, and it must be remembered that in the struggle for existence, through countless years, no one would be here now but for a high degree of this characteristic in his progenitors. It has even been said that it is normal for people to wish that their chief competitors in life were dead, and for the young man to wish a pox or death upon his rival in love. Without entering more deeply into this interesting question, which clearly must involve environmental influences as well as the question of faith, I will only add that, in my opinion, quite apart from his egotism, the murderer is particularly lacking in what the psychologists term " affect tone ". Putting it even more simply, he is completely devoid of that warm human feeling which, with all its faults, is shared, in not such varying degree, by the common mass of humanity. This lack must be specially true of the poisoner, who, nevertheless, possesses a stern fortitude which is in keeping with his grim resolve.

Finally, you will say that I have portrayed for you a seamy and sordid picture of

" the lusts that lure us on, the hates that hound us; our red rags in this patchwork quilt of life ". But if " all the world's a stage and all the men and women merely players ", there must be few who have the opportunity of playing a more important part in the act of life than the doctor, whether for good or ill. Who better than he can feel that though there are treacherous undercurrents on the one hand, on the other " there are waters blown by changing winds to laughter ".

And of those who have been prematurely removed from this transitory sphere by the fell hands of the poisoner, at least we can say:

> Secure from worldly chances and mishaps
> Here lurks no treason, here no envy swells.
> Here grow no damned drugs,
> Here are no storms, no noise,
> But silence and eternal sleep.

So let us leave them, perhaps reflecting that, after all, " the surest poison is Time ".

CAT-SCRATCH DISEASE

(La maladie des griffes du chat)

BY

D. BRYANT, M.B., B.CHIR.

and

R. P. WARIN, M.D., M.R.C.P.

(*Department of Dermatology, United Bristol Hospitals*)

The purpose of this article is to draw attention to a disease which has only recently been recognized in this country. The main features of the condition are described and a probable case arising in the Bristol area is reported.

In 1947, during conversations between Debre of Paris and Foshay of Cincinnati, it became apparent that both these workers had seen numerous examples of this condition during the preceding twenty years (Daniels and MacMurray, 1951). Once its existence was recognized case reports began to appear in the literature, the majority from France and the United States, but also from India (Debre *et al.*, 1950a, 1950b; Mollaret *et al.*, 1950; Daniels and MacMurray, 1951; and Greer and Keefer, 1951. In 1952 an annotation appeared in *The Lancet* and following this cases have been reported in this country (Cox, 1952; Campbell and Wheatson, 1952; and Bettley and Fairburn, 1953).

Daniels and MacMurray (1951) give a full account of the clinical findings in this condition. Many of the cases follow a few days after the scratch or bite of a cat, but there are a large number reported without such a history. At the site of the scratch about half the cases develop an erythematous plaque or nodule on which a vesicle or pustule may appear. Some four to twenty-eight days after the injury a regional adenitis occurs. Commonly one or two lymph nodes are affected but adjacent groups may also become involved, particularly in the neck. The swelling is sometimes quite large (a tangerine and a golf ball are mentioned in the literature) and may become red and tender. Suppuration may occur. The lymphadenopathy slowly settles down over a period of weeks or months.

The patients do not appear to be severely ill, but about half have some fever at the onset. Headaches and malaise are often present. In a few cases a widespread eruption may appear early in the illness. This may be macular or vesicular but erythema multiforme (Greer and Keefer, 1951) erythema nodosum (Cox, 1952) and a papular urticaria-like eruption (Bettley and Fairburn, 1953) have been described.

The blood leucocyte count may show a slight increase in the acute phase but there is no constant abnormality present. Pus, if present, reveals no bacteria and cultures are negative. There is a well-marked reaction to an intradermal injection of an antigen prepared from pus obtained from a lymph node. This takes the form of an indurated papule appearing within forty-eight hours and lasting six to eight weeks. Bettley and Fairburn (1953) have tested the antigen on forty normal adults; three of these showed a positive response but in each case a history of close contact with cats was obtained. Willcox (1952) obtained cross positives in non-specific urethritis but apart from this it would appear that a positive intradermal reaction is good evidence of the disease although of course it would presumably also indicate past infection. The microscopic appearance of the lymph node is not diagnostic but follows a certain pattern. In

mild cases nodules of reticular cell hyperplasia are present. In more advanced cases the centres of the nodules have become necrotic and neutrophil leucocytes mingle with pyknotic debris. The centres of necrosis coalesce into an eosinophilic mass around the borders of which there may be epitheloid cells and giant cells.

A number of conditions have to be considered in the differential diagnosis. The disease is likely to run a more prolonged and less active course than acute bacterial lymphadenitis and there is commonly no neutrophil leucocytosis. On the other hand tuberculosis as a primary focus or as scrofula is a more persistent condition. A tuberculin skin test will be of considerable help (particularly in children). In localities where tularemia occurs this condition may be excluded by agglutination reactions. Lymphogranuloma inguinale may have to be considered and the Frei test is helpful in distinguishing it.

There is no specific treatment. Aureomycin is reported to have been successful in some cases (Debre *et al.*, 1950a) but, in a self-limited condition, assessment of the value of aureomycin must be very difficult. Other antibiotics and sulphonamides are reported as being with effect. The aetiology of the disease is unknown but its general behaviour does suggest a virus infection perhaps of the lymphogranuloma-psittacosis group. Mollaret was able to transmit the disease to one human volunteer out of four, but had more success with apes (Mollaret *et al.*, 1951a and 1951b). Transmission to cats was unsuccessful which suggests that the cat is only a vector.

It is difficult to assess the incidence of the condition. In the past many cases must have been misdiagnosed and it is felt that now that the condition is recognized a number of cases will come to light. Bettley and Fairburn's work would suggest that some 7–8 per cent of adults have had the disease and it may well prove to be a very common benign infection. It is possible that the incidence of the condition has increased in the last few years and this may be due to a habit of closer contact of man with his household pets. It is with the object of drawing attention to the possibility of this infection arising in the West Country that this case report is recorded.

CASE REPORT

A girl of five was brought to the Out-Patient Department with a complaint that there was a spot on the right side of her neck with some enlargement of the glands in the neck. Two to three weeks before the mother had noticed a small pimple on the right side of the child's neck. This had persisted but had not formed a pustule or ulcerated. The child was quite well at the time and her temperature had not been taken.

There was no family history of tuberculosis. The family possessed a male kitten of ten months with which the child played.

On examination there was a small area of erythema, with some induration, behind the right sternomastoid muscle and about two inches above the clavicle. There was enlargement of the right supraclavicular lymph node. This measured about 3 cm. by 2 cm. and was not tender. It was attached deeply but was not fixed to the skin. There was also enlargement of the left submandibular lymph node which showed similar characteristics. No other lymph nodes were enlarged. The spleen and liver were not palpable. The blood leucocyte count was 10,000 (neutrophil 60 per cent). A tuberculin jelly test was negative.

Antigen, specific for cat-scratch disease, was obtained from Dr. Ray Bettley. Intradermal injection of 0·1 ml. of this was followed within twenty-four hours by an area of erythema at the site of injection, which increased to 3 cm. in diameter. One week after the injection a firm, intradermal papule, measuring 1 cm. was palpable. There was no local adenitis. Three weeks after the injection, this papule, though much smaller, was still palpable.

A month after the child's first visit there was little change in the scar on the neck, but the two enlarged lymph nodes were much smaller and were no longer fixed deeply.

SUMMARY

(1) Cat-scratch disease has been recognized as a clinical entity in the past eight years.

(2) The main features of the condition are described.

(3) It is probable that the condition is quite common but not diagnosed as such.

(4) A case arising in the Bristol area is reported.

REFERENCES

Bettley, F., and Fairburn, E. A. (1953). *Lancet*, **1**, 520.
Campbell, E. S. K., and Wheaton, C. E. (1952). *Lancet*, **1**, 975.
Cox, P. J. N. (1952). *Proc. Royl. Soc. Med.* **45**, 456.
Daniels, W. B., and MacMurray, F. G. (1951). *Arch. Int. Med.*, **88**, 736.
Debre, R., Lamy, M., Jammet, M. L., Costil, L., and Mozziconacci, P. (1950a). *Bull. Soc. med. Hôp. Paris*, **66**, 76 (1950a).
Debre *et al.* (1950b). *Semaine Hôp. Paris*, **26**, 1895.
Greer, W. E. R., and Keefer, C. S. (1951). *New Eng. J. Med.*, **244**, 545.
Mollaret, P., Reilly, J., Bastin, R., Tournier, P. *Bull. Soc. med. Hôp. Paris*, **66**, 424 (1950).
Mollaret *et al.* (1951a). *Presse med.*, **59**, 681.
Mollaret *et al.* (1951b). Ibid, **59**, 701.
Willcox, R. R. (1952). *Lancet*, **1**, 470.

SOME NEW METHODS OF RADIOTHERAPY

BY

R. C. TUDWAY, M.B., B.S., B.SC., F.F.R.
Bristol Royal Hospitals

There have been many advances in treatment with X-Rays and radio-active substances. This is an account of those recently adopted and those to be used in future in the Radiotherapy Department at Bristol.

There are two great difficulties in using Deep X-Rays for the treatment of malignant disease. First, " Deep " X-Rays do not penetrate really deeply. Their effective range is a few centimetres deeper than the skin, and it is still impossible with ordinary Deep X-Rays to give an adequate dose to the centre of the pelvis, to lung roots and oesophagus, or even to deeper lymph glands in the supraclavicular region. This is possible, however, by using the X-Rays generated by voltages between two and ten million volts. These rays and machines are called " supervoltage ".

The second difficulty under which the X-Ray therapist labours is that the dose of rays needed to get rid of a growth is commonly rather close to the dose which will excessively damage the normal tissue within the beam. In other words the " therapeutic ratio " is poor. Here also supervoltage rays have a definite advantage because bone and cartilage, which are very easily damaged by ordinary X-Rays, are not so easily damaged by supervoltage X-Rays. In fact all the unwanted reactions of the patient, both skin effects and radiation sickness, are definitely less with supervoltage. Supervoltage machines are, however, very large and costly—for example, a four-million-volt machine (a " linear accelerator ") costs about £40,000.

RADIO-ACTIVE COBALT

Since the atomic energy piles at Harwell and elsewhere have been working, it has been possible to make radio-active cobalt. Radio-active cobalt has one useful property; it emits rays nearly the same as those from a three-million-volt X-Ray machine. This means that if we can get enough radio-active cobalt and enclose it in the middle of a large mass of protecting material, we can get a beam of 2–3 million volt rays simply by leaving a hole in the side of the protecting mass.

Up till now only Canada and the U.S.A. have been able to provide enough radio-active cobalt to give as much power as an actual supervoltage machine. Such a cobalt beam unit from Canada has been presented to Mount Vernon Hospital, Middlesex, and has been installed there.

We in Bristol are now installing a cobalt beam unit, big enough to compete with supervoltage X-Ray machines; it is the first to be made entirely in Great Britain. It should be ready in July this year. At first our unit will not be quite up to the American strength because this country cannot yet provide such a large supply of cobalt but we are expecting that we may have more cobalt later on, and our machine is designed to hold the full amount of 1,500 curies. It is in some ways a handier machine to use as well as being cheaper to maintain, and, in my opinion, it will meet a widely felt need.

Patients with carcinoma within the true pelvis, some with carcinoma of the breast and carcinoma in the head and neck, will be those most likely to benefit from this new machine.

Frequently, after operation for carcinoma of the cervix uteri or after the use of

radium in this disease, Deep X-Rays are necessary to treat the side walls of the pelvis to destroy any deposits left there. This has never been easy to do with ordinary Deep X-Rays. The cobalt beam will not only give a much better dose to these parts but will cause less damage to the skin, bone will no longer be an obstacle, and the patient will have less constitutional upset. We can now give a dose which may destroy a majority of these deposits of growth instead of only a few of those which are very radio-sensitive.

The amount of Deep X-Rays for carcinoma around the larynx is usually limited by the fact that perichondritis is caused by a dose lower than that which will burn soft tissues. Carcinoma of the larynx must therefore be given a lower dose of Deep X-Rays than the same type of growth in other sites. Cartilage, however, has little more sensitivity to rays from the cobalt unit, so with this unit a better dose can be given to the larynx. Most growths of the jaws and sinuses involve bone or are surrounded by bone. These also are better treated by the cobalt beam because bone is another tissue which is selectively damaged by Deep X-Rays and is an obstacle to them; but the cobalt beam treats bone in the same way as soft tissue, and therefore has none of these disadvantages. Likewise the bone of the skull will not absorb rays from the cobalt unit intended for intracranial tumours as it would absorb Deep X-Rays.

We have had to face some difficulties in the construction of the machine, and have now been working on the project for two years. The cobalt is very radio-active indeed, and must lie in the centre of a cylinder of lead two feet in diameter, otherwise the whole of the patient and the staff of the department would be irradiated. No individual can approach within a yard of the unprotected source for more than a few seconds without receiving a dose which could be harmful. A two-foot-diameter mass of lead is very heavy and unwieldy, so we are using uranium metal instead of lead. The uranium cylinder need only be eleven inches in diameter.

One advantage of uranium is that the units can be supported on commercially built stands like those used for Deep X-Ray machines, because they only weigh 600 pounds each, whereas the lead protected unit weighs over 2,000 pounds and must have a specially built support. The units are raised, lowered and rotated for the aiming of the beams by means of electric motors in the supporting stands.

When the shutter is open and the beam is in use, it is essential to prevent rays from escaping from the room. This means that the walls of the room must be of a thickness varying from eighteen inches to three feet. This has presented a major problem in the Bristol General Hospital where there is no room to build anew, and such heavy new walls could only be supported at ground level. A solution has been found in the arched cellars which support the internal courtyard of the building. These arches have walls about two and a half feet thick which form an excellent protective surround to the treatment rooms. As can well be imagined these cellars in their crude state are not very inviting for the treatment of patients. The major work of the whole project is the heating, ventilation, lighting and decorating of these rooms. Doors to prevent escape of rays would have to be very heavy—equivalent to concrete eighteen inches thick. Fortunately the rays have very little ability to turn corners so their exit from the rooms can be blocked by having a twisting passage with thick walls for the entrance. The provision of such tortuous entrances to the rooms has meant careful planning to fit them within the existing walls. In other places doors like those of ordinary X-Ray rooms are also being used. The electrical controls can then be so arranged that anyone who accidentally enters the room while the unit is in action will automatically turn it off.

The cobalt unit which is being installed at the Bristol General Hospital is a double unit, with two separate parts in two adjacent rooms so that two patients will be treated at the same time. This has been done to minimize the main disadvantage of radio-active cobalt which is that the radio-activity gradually fades away and after five years only half the strength is left. This means that after about four years it will be too weak to treat the deepest tumours and must be renewed. The cobalt in one treatment room will be used for these deep tumours for four years. Then it will be transferred

to the other room for a further four years where it will be used for the treatment of more accessible tumours which are sensitive to lower doses.

Radio-active cobalt can also be used to replace radium in needles. It has the advantage that, unlike radium, it does not need to be kept in a platinum needle. The cobalt needles can be made of stainless steel which is much stronger than platinum. We have made them at the Bristol General Hospital, and we find them very satisfactory. The action of these needles on the patient and on the growth is indistinguishable from that of radium. Loss of a cobalt needle is less serious than that of radium.

RADIO-ACTIVE CAESIUM

Before long, radio-active caesium should be available. This substance is one of the waste products from the atomic piles. It cannot be used in an impure state, but if a purification plant is set up it can be made available in large quantities. It emits rays which are not quite so powerful as those from cobalt; they are nearly equal to those from a one-million-volt X-Ray machine.

RADIO-ACTIVE IRIDIUM

We are also using radio-active iridium in carcinoma of the cervix uteri. The reason for this is that the uterus and vagina tolerate radium extremely well and can take a dose heavy enough to reach the lymph glands of the side wall of the pelvis. Unfortunately the bladder and rectum are much more sensitive structures. Attempts have been made to introduce screens of platinum between the radium and the rectum, but unfortunately the rays from radium are so penetrating that very heavy and bulky screens must be used. The rays of radio-active iridium penetrate the tissues easily, but are still not too energetic to be stopped by a small screen, especially if the latter is made of uranium.

SYSTEMIC USE OF RADIO-ACTIVE ISOTOPES

In the last year or two, there have been no great advances in the systemic use of radio-active isotopes; but methods have been improved.

The use of radio-active iodine in thyroid disease is now well established. Carcinoma of the thyroid may sometimes take up radio-active iodine and enough may be concentrated to destroy the growth. Unfortunately the normal thyroid tissue tends to absorb the greater part of the administered iodine; so the first step in this treatment is usually to remove the normal thyroid. Occasionally the tumour tissue absorbs iodine so actively that this step is not necessary. We have successfully treated one such case. This patient's growth had recurred after both surgery and Deep X-Rays. A single large dose of radio-active iodine was then given, the swelling disappeared and she has now been free of recurrence for two years.

THYROTOXICOSIS

Radio-active iodine is of value in those cases of toxicosis in which surgery is unsafe, and in which the thiouracil group of drugs is not tolerated or has lost effect. In primary Graves' disease it is usually possible to achieve a euthyroid state with radio-active iodine, though there is always some risk of leaving the patient somewhat myxoedematous. In secondary thyrotoxicosis from nodular goitres the correct dosage is very difficult to judge and over-treatment with consequent myxoedema is quite common. However, this may be helpful because the patients with secondary thyrotoxicosis often have some heart failure, and the sub-thyroid state is an advantage. In our experience the treatment of these secondary toxic goitres with radio-active iodine has been very successful. So far we have treated about a hundred patients and our results will soon be published.

HEART DISEASE

In some cases of heart disease not due to thyrotoxicosis, radio-active iodine is given deliberately to produce some myxoedema and so relieve the work of the heart. The iodine is given by mouth. It produces no reactions apart from slight nausea.

RADIO-ACTIVE PHOSPHORUS

Systemic radio-active phosphorus has been tried for the treatment of malignant disease, and rapidly growing tissue, such as a tumour, will take up more phosphorus than normal tissues. The results however are not good. Phosphorus is stored in bone; therefore tumours originating within a bone may be treated if they are sufficiently sensitive. Both myeloid and lymphatic leukaemia respond well for a while. Other reticuloses, of which mycosis fungoides is one, sometimes respond well. In multiple myelomatosis there may be considerable, though temporary, relief.

FUTURE RESEARCH

Little is known about what makes a tumour sensitive to radiation. At the moment various lines of research are in hand which include increasing oxygen uptake by a tumour during radiotherapy, and administration of vitamin K. Neither approach has proved promising so far.

Other work is already needed to help us select those patients most likely to benefit from radiotherapy, for histological appearances are a poor guide and we have often to resort to trial and error.

Progress in radiotherapy is rapid and before long we may have the answers to some of these outstanding problems.

"GENERAL PRACTICE"*

BY

G. F. ABERCROMBIE, V.R.D., M.D.

(*Chairman of Council, College of General Practitioners*)

Mr. President, Ladies and Gentlemen,

I must first thank you very sincerely for a most flattering invitation to address this famous Society. I have undertaken it with some misgiving, for it is not easy to present a theme from general practice suited to your varied membership. I can only hope that some reflections on medical education may prove of interest to you, and I shall try to indicate what I have found of greatest value.

[Dr. Abercrombie then recapitulated one of those unfortunate cases, recently reported in the Press, in which a mistake in diagnosis combined with some lack of co-operation between hospital and practitioner had led to a tragedy. He continued:]

Here let me implore all general practitioners to follow their cases into hospital. Particularly is this desirable if the patient is a child. Never let us forget that when a patient goes into hospital she leaves outside her only real medical friend and adviser. A visit from such a one is an enormous comfort and encouragement, and if we feel that what we thought necessary has not been done, we can make the opportunity to seek out the man in charge and discuss the case with him.

Not only that, but we can explain to the patient what the hospital is doing. It is a very common complaint that "they tell you nothing in hospital". The general practitioner then, sees the patients early, may see them as often as he wishes, may see them in their own homes, and should follow them into hospital to encourage and to explain.

Education, you will remember, has been defined as the casting of imaginary pearls before real swine, and as I pause to survey the long road so laboriously travelled, I find much force in that definition. Far away on the distant horizon are some low foothills, the preclinical subjects.

I may be wrong, but I am not conscious of being of inferior value to patients, because I now remember absolutely nothing of the anatomical structure of the skull of the dolphin, or of the reproductive arrangements of the earthworm; but I do very much regret that no one ever demonstrated to me the brain of the bird. It would have been, I think, very salutary for a medical student, at the stage when he had been immersed in the theory of man's evolution, to have been made to realize that, though apparently the most successful, the human brain is *not* Nature's last word on the subject. The avian brain is a later experiment and there may be others yet to come.

Of all that one was taught in those far-off days lamentably little now remains, but there are some scenes, not of detail, but of men, which have never been dimmed by the passing of years—scenes which, like beacon fires on mountain tops, have illumined some general principles of life-long value.

Having passed Anatomy and Physiology, three of us worked for some months as unqualified assistants to the Resident House Surgeon at Addenbrookes Hospital, Cambridge, and into our room one day strode Carless, already of course known to us by repute as part-author of the *Textbook of Surgery*, Rose and Carless, and I suppose in his day one of the most famous surgeons in England. He had come to operate on a soldier, whose sciatic nerve had been severed in mid-thigh, and he asked whether he might borrow our *Gray's Anatomy* for a moment to look up the relations of the

* A paper read to Bristol Medico-Chirurgical Society on 14th April, 1954.

sciatic nerve. I was never more astonished in my life—" Look up the relations of the sciatic nerve! " " Good God," I thought, " Doesn't he know them? " " Ah yes," he said, studying the diagram, " I see," and led the way to the theatre. What followed was to us intensely dramatic, an eighteen-inch incision down the back of the thigh, one other deft movement, and there it was. Now, if a master of surgery of that calibre was not too proud to revise his anatomy at a critical moment, surely we might follow his example, and certainly, from that day to this, I have never hesitated to look up anything.

Is it really necessary, I wonder, for every single one of us to make the tremendous intellectual effort to learn the whole topographical anatomy by heart? As those who aspire to be surgeons are nowadays compelled to return to the study of anatomy for the Primary Fellowship, would it not be reasonable to lessen the anatomical burden before qualification? And would not the same line of argument hold for biochemistry and physiology?

A little later, I was profoundly impressed by the physician, Drysdale of St. Bartholomew's in London, who in a very few words precisely defined the ideal doctor-patient relationship. " Gentlemen," he said, and he shook a finger at us, " you are not to suppose that you are sent into this world to order people's lives. You don't give orders; you give advice." No more valuable admonition could have been addressed to those who perhaps already were beginning to think they knew something, were somebody, and ought to be obeyed. " You don't give orders, you give advice." How exactly true that is. All our lives we are in effect saying to patients: " I advise you to stay in bed two more days; I advise you to have that wrist X-rayed; I advise you never to run for a bus "; and so on. There must not be any attempt at compulsion, and there ought not to be any attempt at argument. An argument between a doctor and his patient is generally undignified, seldom successful, and often leads to such loss of mutual trust and confidence that complete rupture follows. Much of the art of general practice lies in the management of one's fellowmen, and to secure the willing acceptance of unwelcome advice needs tact and skill.

What sort of answers, do you suppose, would be given by a student in his final examination, to questions such as these? It would be made clear of course, in a footnote, that no marks would be given for throwing up the case and running away.

1. You are called to a boy, two years old, and after careful consideration of the history of his illness and of the physical signs you are convinced that he has an intussusception. You are also convinced, from your previous knowledge of his parents, that at the mere mention of the word " operation " they will panic and dismiss you. How do you proceed?

2. In response to an urgent request received at 3.30 a.m. on a Boxing Day, you go to see a woman of thirty-eight, and although the way she rolls about in bed with abdominal pain is most unconvincing, you nevertheless form the opinion that it is an acute abdomen. You advise observation in a nursing-home, but this advice proves unacceptable to her and to her husband, who turn out to be Christian Scientists. What do you do now?

I had the good fortune to dress for and later to be House Surgeon to Sir Holburt Waring. What did he teach which has proved of value in the years of general practice? Certainly not surgery, for apart from a few well-worn formulae of which " Hernia, inguinal, right, indirect, epiplo enterocele, reducible, recurrent, non-strangulated " was a characteristic example, he appeared not to teach any scientific surgery at all. But Waring was a great man. He taught us to observe and he taught us to record, and any attempt at hedging was instantly pilloried. His most valuable contribution to me personally was his abhorrence of the word " slight ", which he would never allow to be used. " Slight oedema," " Slight dulness," " Slight tenderness "— these were OUT. Either the feet were swollen, or they were not; there was dulness over the base or there was not; the belly was tender or it was not; and we were absolutely forced to say and write one thing or the other. Not very long ago, I received from the radiological department of a hospital a six-line report, which contained the adjective " slight " three times. It was not of the slightest use.

Now this habit of making up one's mind and coming to a decision has been of very great help. Is this belly tender or is it not? If it is not, almost certainly all is well; but if it is, almost certainly it ought to be explored. I have practically given up the attempt at brilliant differential diagnoses in the acute abdomen, fascinating though such speculations are. I don't think the general practitioner is required to distinguish between, say, a ruptured ectopic gestation and a twisted ovarian cyst, or even between an acute appendix and a perforated peptic ulcer, but he *is* required to decide whether it is SAFE to leave such a patient in her own home throughout a long night. I entirely agree with the argument of a country practitioner that in such cases one ought to come to a firm decision on the spot at the first visit.

For a final illustration of this kind of instruction I must return to Cambridge to sit at the feet of Sir Clifford Albutt. " If I had my way," wrote this wise and humane physician, " no man, not even a pauper, should die of acute or obscure disease without a consultation." This plea is possibly less cogent now than when he first uttered it in 1889, but it is a constantly recurring situation of which inexperienced practitioners ought to be forewarned. Who is there among us who has not suddenly realized that the apparently trivial illness has unexpectedly taken a grave turn, or that, after two days in attendance, one must admit, if one is honest, that one really does not know what the diagnosis is? It has been said, too, that one cannot hope to diagnose a condition one has never seen and I assure you that I see, almost every month or two, something of which I have no previous experience, and which I am never likely to see again in private practice.

On too many occasions, I fear, the benefit of a second opinion, which Albutt so strongly advocates, is a good deal less than it should be. I sometimes think that those who offer these opinions in acute or obscure disease under-estimate the good sense of those who receive them. Robert Birley, now Headmaster of Eton, once remarked that, " if there is anything in the educational tradition of Western Europe, derived from the great teachers of Athens, the honest penetrating study of great literature or philosophy, or mathematics or science, should produce men of integrity, who can appreciate nonsense when they meet it." Now to give two examples: I may tell you, what you will hardly credit, that I was offered the diagnosis of " streptococcal bronchorrhoea " for a man who was coughing up the contents of an abscess of the lung; and, for a woman who died of multiple new growths of the small intestine, " fatty diarrhoea with constipation ", was suggested. One of the subsidiary functions of the family doctor is to know when the consultant is bluffing. It is of course never easy to say " I don't know ", but it can be done and sometimes it ought to be done. I recently summoned a paediatrician to see a baby who was causing me some anxiety. After a thorough examination, he discussed the possibilities with me, and then turned to the mother and with disarming and quite admirable frankness, admitted that he could not make a diagnosis. " Your doctor has called me because he cannot put his finger on the exact spot of this trouble and *I am in the same position.* We ought to take him into a ward in the Children's Hospital, and investigate the urine, the cerebrospinal fluid and so on."

Now that, if I may say so, is consultation at its best, and I have to add with very great regret that the National Health Service is killing it. In former days the consultant made his way largely by the good will and the good opinion of the general practitioner. But now he is independent, for his income is assured, and in consequence he is rapidly becoming an absolute autocrat. He is not to be found at the week-end, his night-work is done by his registrar and his houseman has instructions not to admit certain types of case from an Emergency Bed Service. This is not the occasion to discuss the allocation of hospital beds, but I cannot believe that conduct of that kind is a good example to students.

Some people will tell you that general practice is 90 per cent trivialities. I've never found it as dull as that. It depends entirely where your interest lies. If you are interested only in tonsils or hernias, leucocytes or spirochaetes, you will miss the heart of it, for general practice is concerned with men, women and children. If then you

are interested in humanity, you will find it fascinating, though full of strange surprises, of which perhaps the student ought to be warned so that he may keep a straight face.

If, for instance, he should find an old lady sitting up in bed, and with a large pair of scissors cutting carefully along the lines of perforation in a roll of toilet paper, he must *not* say, " Madam, that material can easily be obtained in separate sheets, if you so desire." No, no, for what he witnesses is the newest branch of scientific medicine—Occupational Therapy.

Many years ago, I was asked to see at frequent intervals and at a considerable distance an old bankrupt dying of cancer and I was implored *not* to disclose to him the nature of his illness, of which, happily, he was ignorant. I did the best I could, but this assignment proved too difficult for me in my youth, and after two or three weeks the old man suddenly said, " Doctor, we're not making progress. I think I'd like a change." What could I say? Another man took my place, the patient died three days later. That kind of treatment is certainly hard, but one has to learn not to be hurt.

There is, of course, the other side of the picture. All successful doctors, as you know, sport a gold fountain-pen. Mine came to me as follows: For several days I missed the diagnosis of acute appendicitis in a stout Jewess, aged about fifty, in consequence of which the appendix perforated and an abscess formed which had to be drained; after that two sinuses persisted and a second operation had to be done about ten weeks later. Her illness in fact lasted nearly five months before her husband, in great gratitude, presented me with a remarkably fine gold pen, which was duly stolen from me by an Irish housemaid. However, the insurance company provided a poor substitute, which I have used ever since, and I still attend that old lady and we are very firm friends.

I think the young man who goes into general practice would appreciate a friendly warning that much disappointment lies ahead of him, and would welcome some advice as to how to make the best of it. Consider, here he is, eager, willing and sufficiently skilled to do quite a number of difficult things. He can remove an appendix, or a pair of tonsils; he can repair a hernia; he has probably had a good experience of the use of plaster of Paris in the treatment of fractures. He has learnt to put a hollow needle into the spinal theca and other tubes and cavities and perhaps into joints. I always like the old story of the two house physicians who had attempted to tap the pericardium, and considered that the fluid they withdrew was very deeply blood-stained. He can prepare with his own hand a tolerably good miscroscopic section; he may have mastered the technique of blood sugar and similar estimations, but when he gets into the ruck of general practice he will soon find it impossible to keep his hand in at everything. Suitable cases, so frequent in hospital, are uncommon in practice, and the care of equipment, always at hand in hospital and cleaned up and put away afterwards by someone else, takes more time than can be spared. And so he finds that, keen though he is, he has to let much of the varied and interesting work go away from him.

The best compensation is to choose some strictly limited field, which particularly appeals to him, put in half a day a week at it as a clinical assistant in hospital, and keep that going in his own practice.

[After discussing some of the problems of obstetrics Dr. Abercrombie turned to the subject of neurosis.]

This meant in those days—I am not sure what it means now—that if no adequate hypothesis could be found to fit the patient's symptoms no organic disease existed. I can see, in my mind's eye, an old man in my hospital whose prostate had been removed. After his operation he vomited over and over again. At first this was attributed to the anaesthetic, but after several days that diagnosis had to be abandoned and no other offering he was called " neurotic ". Then he died. " How extraordinary," I thought, as I made my way to the post-mortem room, "that a man can actually die of a neurosis." And there, of course, as so often happens, the truth came

out and the organic disease was demonstrated—in that case an obstruction by a distended oesophageal pouch.

That kind of thing will happen frequently unless one makes a rule, when confronted with persistent symptoms with or without physical signs, not to lose sight of the patient until a satisfactory explanation is forthcoming.

Let me illustrate this by recounting one of my own failures. On May 26th, in a certain year, I saw for the first time a man of thirty-eight who had been complaining of pain in his left shoulder for eight months. Novocain injections had given temporary relief and infra-red radiation would relieve him for half an hour. When severe, his pain spread to the arms and forearms, up the neck behind the ear and to the top of his head. It was not provoked by movement or exertion, but might come on when he was resting or during sleep. He had been passed for life assurance and I could find nothing the matter with him. I got him X-rayed. There was no evidence of any bone injury, disease or displacement in the shoulder, and no abnormal shadows in the soft tissues. There was no sign of any bony cervical rib and no evidence of any bony arthritic changes in the cervical spine. A masseuse helped him a great deal, but on July 6th—six weeks later—he still had two symptoms: (1) pain on the left side of the neck and up to the crown of the head when he lay down in bed at night, which might last half an hour or more before he fell asleep; and (2) a throbbing down the back of the left arm and into the palm of the hand via the inner side, coming on about 10 a.m. and lasting, if not relieved by veganin, several hours. Being completely stumped I took him to the best neurologist I know. This consultant confessed himself entirely unable to solve this problem in the absence of any physical sign to localize his symptoms, that is, any change in reflexes, any weakness, any wasting and so on. He might, he said, have a neuroma somewhere but he did not think lipiodo injection was really justified at the moment. Now for me, at any rate, this is the danger point. If I do not take a grip of myself, I am liable to begin to think he is a neurotic, and once I have labelled a patient " neurotic " I find it almost impossible to remain in sympathy with him, and to treat him as a reasonable being. No matter what he says, one still thinks, " Oh, he's only a neurotic ", and the number and gravity of mistakes which can be made in that way is appalling. However, they are not mistakes which can be overtaken and corrected. One has practically told the man there is nothing the matter with him and that it is all imagination. How is that going to be explained away when the truth is out?

Fortunately for my peace of mind I did not label this man a neurotic, for within two months he too was dead. A few days after the consultation with the neurologist, he took a little rowing exercise on the lake in Regent's Park, and a lump appeared in the middle of his right thigh towards the inner side. This gradually increased in size, and when he showed it to me on August 4th, it was an obivous aneurysm several inches in diameter. In hospital an X-ray of his chest revealed a large aneurysm of the aorta as well, which progressed rapidly. His Wassermann was positive, but massive anti-syphilitic treatment throughout failed. Finally the aneurysm in his thigh burst and he died the next day.

I have told you this long story to emphasize two points: first, if you cannot make a diagnosis, don't lose sight of the patient, keep him on the list, see him again and again, for something is sure to turn up sooner or later; and, second, don't label him neurotic and lose all interest and sympathy.

The recognition of early cases of cancer is immensely important, as you all know, and yet I find it one of the most difficult sides of our work. The public are easily frightened, which hampers us, for we are reluctant to cause unnecessary alarm and perhaps precipitate good people into anxiety states. Secondly, there is nearly always some apparently most cogent reason why this particular patient should not have cancer. Perhaps he has had this kind of flatulent dyspepsia for twenty years; perhaps his abdomen has been recently explored for some other condition, and we feel we know all about him. I can show you what I mean by two recent cases of carcinoma which illustrate the remarkable red herrings liable to be drawn across the path of

the right diagnosis, and above all, demonstrate that when we take over a patient, we must never take over the previous diagnosis as well, until we have confirmed it for ourselves. No matter what other people have said, and no matter how eminent or distinguished they may be, it won't do to take their word for it. We must investigate for ourselves. There may be something else.

The first was a doctor, aged about sixty, formerly a psychiatrist, but now largely disabled by disseminated sclerosis, which he had had for many years. He was incontinent of urine and incidentally had been under the care of a surgeon, who had tunnelled his prostate and had been keeping him under regular observation. I was asked to go and see him about once a month and to let him talk. When one is young, one is inclined to think that that kind of visit is a complete waste of time and isn't really Medicine at all; but as one grows older one comes to realize that, to many people almost cut off from the rest of the world by some infirmity, the occasional visit by one of their remaining friends, in this case their doctor, is one of the few pleasures left to them. " I can't talk to Mr. So-and-So ", said a widow, referring to a consulting physician. " Why not," I asked, " he's a very charming man." " Yes, I know," she said, " but he always asks questions, and although I give him all the answers I don't feel I've *talked* to him."

One day my psychiatric friend said he was having a little trouble with his sphincter ani. Now, if there is disseminated sclerosis, it is very likely indeed that there will be trouble with the sphincters, and as I've told you, he had no bladder control at all. I naturally assumed that control of his bowel was affected by the same cause, but just to reassure him, I did actually put a finger into his rectum, and there to my horror, I felt a thing sticking into the lumen about the same size and shape as a cervix uteri, but very hard indeed. That man and his wife are very grateful, but I can confess to you, that we were all very fortunate.

About two years ago a colleague, who avoids obstetrics, asked me to attend a patient of his, whose second baby was due in a month or two. She was thirty-nine and after one child had had several miscarriages. This pregnancy therefore was probably her last chance of having another baby. She developed some piles towards the end of her time. She had had them before, she said, but had never bothered about them. During the first stage of labour the rectum was turned almost inside-out by pressure, and was pushed back from time to time to relieve the congestion. All went well and when she was fit to get up, I sent her back to her own doctor, told him the story, and said she really must have something done about her piles. He didn't examine her himself, but sent her straight on to a specialist, who reported that she had an annular carcinoma and advised an abdominoperineal. We had been so hypnotized by the confinement that this possibility had never entered our minds. Perhaps it was just as well, for what we should have done if we had discovered it in the last few weeks of pregnancy, Heaven only knows.

As to prognosis, I believe that that cannot be learned in hospital, and that only a long experience of sick people in general practice can give any knowledge of it. It is astonishing how long patients with a mortal illness can survive in comparative comfort, when in the care of skilled nurses and devoted relatives. The pinning or plating of a fractured femur in the elderly is now a commonplace and will generally save much suffering, but it is perhaps not so often realized that the wearing of a truss for many years adds greatly to the burden of old age. I do unhesitatingly advise a surgical opinion in such cases, even when the circumstances are obviously unfavourable.

I still find it extremely difficult to appreciate day-to-day or week-by-week changes in a patient's progress towards recovery or otherwise. When I began to practise and found a marked change or a new physical sign at a second examination, I could not help feeling this must be something which I had previously missed. This of course is most disconcerting and it has taken me many years to realize that the rate of change in signs can be almost unbelievably swift.

I wish that as a student, I had been initiated into some sort of Nosological Record, so that I might have kept as I went along lists of, say, the incidence and outcome of

various types of malignant disease; the varieties of minor fractures, the success or otherwise of operations on the knee joint or the gall bladder or the prostate, and other interesting phenomena. I wish, too, that when I was a houseman, I could have seen something of the planning and organization which goes on in a big hospital. It would be no bad thing if housemen were taken by their chiefs, once in a while, to a meeting of the Council or Finance Committee or similar body. We all rise to positions of responsibility in one field or another and we ought to be introduced to this side of medical life, while we are still young. It would, I am sure, be a most stimulating and enlightening experience to listen to the formulation of the plan for a new wing or a new department, to watch an able chairman control an acrimonious discussion, or to hear a harrassed treasurer describe his financial resources.

In conclusion, Sir—and I must thank you for having been so patient with me—in conclusion I wish that, when I first entered general practice, there had been a College of General Practitioners. How eagerly one would have joined it! How much it might have accomplished in all these years!

Still, better late than never. We have made a start.

PLATE XVIII

Coronal section of brain showing cyst in 3rd ventricle

Photomicrograph of cyst wall with colloid contents (large spaces in colloid are fixation artefacts). × 90

COLLOID CYST OF 3rd VENTRICLE*

(A Clinico-Pathological Report)

Dr. W. O. Spence: The patient was a Hungarian and he was a very fit and strong man. He was working as a carpenter although he was actually a horticultural expert. He told me that he had been having headaches for about 6 months before his death in December, 1953, and he had not taken very much notice of them but that they were getting worse. The first intimation I had that anything was wrong was when he came to me complaining of headache and saying that the light bothered him: he was very photophobic. Two days later I was sent for because he had a very severe headache and vomiting. I thought it might be a sub-arachnoid haemorrhage and that it was sufficiently serious to be investigated further, so I sent him to Ham Green Hospital. He was there for two days and was sent home pending further investigations without any definite diagnosis being made but with the very interesting finding of changes in the right fundus: that is, early swelling of the disc. The left fundus was normal. He had recovered completely, resumed his occupation and attended hospital as an outpatient for further investigation. He continued to have headaches and occasional vomiting. I saw him on the 25th November when he had a severe headache and could not stand the light. His headache increased and eventually he was admitted to the Bristol Royal Infirmary. He was quite rational; blood pressure was normal. His disc was still showing optic atrophy and he died after six or eight hours.

Dr. G. D. W. McKendrick: I must comment on something I have just learnt from the history. This man denied continuously that he had ever had a headache before the 28th October, and as far as I was concerned his symptoms started four days before he came into Ham Green Hospital.

On admission he had a temperature of 99·8° F. which was the only fever recorded whilst in hospital. There were no abnormal physical signs on examination apart from the right disc which was a little pink with some blurring of the upper and lower margins; the blood pressure was 120/80, the cerebro-spinal fluid normal with 4 cells and 20 mgm. per cent. protein. The pressure was not measured.

The day after admission he was much better and his headache had gone in forty-eight hours. Apart from one small vomit after admission he remained well and did not develop any focal C.N.S. signs. He was very loath to stay in hospital and was sent home with a tentative diagnosis of optic neuritis. He attended Out-patients on the 9th December, when he felt quite well. He still had no focal signs but his right disc was the same. However, he had had one further attack of headache and vomiting which started about a week after he left hospital and lasted ten days. This suggested that his disc changes were due to raised intracranial pressure rather than to optic neuritis, and I referred him to Dr. Campbell.

Dr. A. M. G. Campbell: He never got to me. The first time I saw him was on the post-mortem table. Dr. Colson can tell you more than I can.

Dr. G. Colson: I can just confirm that he was finally admitted to the B.R.I. on the 13th December, when he was perfectly orientated and stated that he had had these attacks of headache for six weeks. The story here is absolutely as described before. He had had one attack since leaving Ham Green Hospital.

In the ward, Dr. Bain noted that he was a strong man, with marked headache, restlessness and photophobia. There were no abnormal physical signs whatever apart from the right optic fundus; there was no papilloedema, but only some congestion of the vessels. The left fundus was normal. He was thought to have migraine and was

* A case report at a Clinico-Pathological Conference held on 2nd March, 1954, by Prof. T. F. Hewer, in the Department of Pathology, University of Bristol.

given analgesics. A few hours later he complained of headache, vomited three times and suddenly died.

Professor C. Bruce Perry: Was his optic atrophy primary or secondary?

Dr. G. Colson: Swelling and papilloedema and optic atrophy are mentioned here.

Professor Perry: Can anybody tell us what the onset of the headaches was?

Dr. Colson: They were gradual.

Professor Perry: How long did they take to work up to maximum?

Dr. Colson: The headache started on the 28th October and lasted until the 31st October; he vomited at work and went home. I never saw him with severe headache. The position of this headache was frontal, slightly more marked on the right side. He never lost consciousness.

Professor T. F. Hewer: Dr. Farrant will now give us the post-mortem findings.

Dr. P. C. Farrant: This man was extremely well developed and muscular. I noted that his left pupil was larger than his right. The brain weighed 1,545 grams, which is a bit large; the convolutions were flattened out and, looking at the base of the brain, there was grooving at the bottom of the cerebellum which indicated that some coning had taken place and both tonsils of the cerebellum had been pushed down through the floor of the foramen magnum. There was also slight protrusion of the 3rd ventricle. So here was a rather large and flabby brain. At this point, I asked Dr. Bishton's advice as to whether I should cut it or not. Dr. Bishton took one look at the brain and said that I should make one cut—it might be a 3rd ventricle cyst: it was! There was a colloid cyst of the 3rd ventricle which had been compressing the foramina of Monro laterally causing dilation of both lateral ventricles and hydrocephalus. It arose from a wide but rather tenuous attachment from the roof of the 3rd ventricle: histologically this cyst is filled with colloidal material which before fixation was translucent, and in the colloid material there are scattered groups of inflammatory cells, leucocytes and a few macrophages. The inner lining has a single layer of cuboidal epithelium, in places ciliated. Externally the cyst is lined by rather flattened cells which may be a definite layer or may be due to stretching.

These cysts have been variously described as arising from the choroid plexus, the ependyma or the paraphysis. They only occur in the 3rd ventricle and this fact makes their derivation from choroid plexus and ependyma less likely. On the other hand, choroid plexus during development does grow down from this position to the lateral and the 3rd ventricles. As to the theory that they come from the paraphysis, this is a structure found in certain lower vertebrates—frogs, some fishes and prominently, I believe, in the sturgeon. It is an out-pouching from the front of the 3rd ventricle lined by a derivative of ependyma. In man it is a transient structure which is present only while the embryo is from 19–32 mm. in length. Histology of this particular cyst does not definitely decide the matter but ciliated epithelium is recognizable and the most likely origin is from the paraphysis.

There was another incidental finding: the spleen, thymus and left tonsil were enlarged and I thought it possible that we might be dealing with a case of generalized lymphoid hyperplasia. The lymph nodes, however, were not enlarged. The enlargement of the spleen was due entirely to gross acute congestion. The thymus showed reactive hyperplasia and was, in addition, fatty: it is possible that this could be its normal state in some young healthy adults—this man was 29. The tonsillar enlargement was due to the presence of several hard accretions in its crypts.

So there it is: a case of colloid cyst of the 3rd ventricle with blockage of the foramina of Monro, the development of acute hydrocephalus and death.

Mr. D. G. Phillips showed a ventriculogram from another patient which revealed a colloid cyst which was removed successfully and he also showed the cyst itself:

"The cyst is quite simple to remove, approaching it through an opening made in the frontal lobe.

"Alternatively the cyst may be simply coagulated to avoid damaging the large veins in the neighbourhood of the foramina of Monro which might cause severe haemorrhage or infarction.

"While the syndrome of intermittent paroxysmal headache is characteristic, diagnosis on clinical grounds alone is not often made correctly; the ventriculography appearances, however, are pathognomonic."

Dr. Campbell: I have little to add except to say that one could not tell on the clinica story this was a 3rd ventricle cyst. There was no temporary weakness of the legs: no focal signs. Why was there a chronic swelling of the disc over this period? There was no high pressure; the disc swelling is confusing.

Dr. Farrant: I took the right eye out and on section there was no abnormality in the optic nerve or retina.

SPARE COPIES WANTED

Medical Journal of the South-West

Vol. 70 (i) 1953, No. 253

The editor would be glad to receive a number of copies of this issue to complete several exchanges for foreign journals.

If you can spare your copy of this issue, please send it to Mr. A. E. Roberts, Medical Librarian, University of Bristol, or leave it at the B.R.I. for the editor.

Correspondence

Memorial to the late Dr. Norman Lloyd Price

Dr. Norman Lloyd Price was Honorary Physician to Out Patients at the Bristol Royal Hospital for Sick Children from 1937 and the first Shaw Lecturer in Diseases of Children at Bristol University until his death in 1945. He was keenly interested in the development of paediatric services in Bristol, and had looked forward with great enthusiasm to the establishment of the University Department of Child Health and during the short time available to him, he made a very substantial contribution to the practice and teaching of paediatrics in the City.

About six years ago we opened a fund to provide a memorial to him and it seemed appropriate that this should be devoted to some purpose which would further the development of his chosen branch of medicine. We originally suggested that the memorial should take the form of a prize in Child Health for undergraduates at Bristol University. However, since subscriptions were originally invited, the University has received other endowments for this purpose. After discussion it was decided, therefore, that the most fitting memorial would be an endowment for the purchase of books for the section of Child Health and Paediatrics in the University Medical Library. This would benefit a large number of students and practitioners in Bristol and the South West.

Many friends, relatives and members of the Medical and Nursing professions have sent contributions so that recently we were able to give to the University the sum of £300. This has been very gratefully accepted by the Council and the income from this endowment will be used for the purchase of books for the medical library; each will bear an inscription stating that it has been purchased from the Norman Lloyd Price Memorial Fund.

BERYL D. CORNER.
ARTHUR L. EYRE-BROOK.
FRANCIS J. W. LEWIS.

Annotations

EXTENSIONS TO THE UNIVERSITY DEPARTMENT OF MEDICINE

During the winter there were important extensions to the University Department of Medicine. The cramped attic-rooms at the top of the Infirmary (which had been used for occupational therapy) were demolished and replaced by a well-equipped lecture theatre. This theatre was sorely needed. It has been designed not only for lectures but for clinical demonstrations and film projections. It adds much to the amenities for undergraduate and post-graduate education.

There are several other new rooms suitable for use either as separate laboratories or private offices.

TOO MANY MEDICO-SOCIAL WORKERS?

We are all familiar with the increasing number of specialties within the ranks of the medical profession; indeed, it is becoming quite difficult to keep track of the many different " -ologists " and " -icians " who minister to our present-day needs. What may not be quite so well known is that a parallel development has been going on in the medico-social field.

Dr. R. C. Wofinden, in a recent article,[1] published in *The Medical Officer*, draws our attention to the wide variety of medico-social workers. He found that there were no less than thirty-five different medico-social workers, visiting the homes of Bristol people.

The author conducted a pilot survey on approximately a thousand families who were being visited in their homes by health visitors. The sample was heavily biased by families with mothers between the ages 20–45 and therefore firm conclusions may not be drawn from the findings. Nearly 14 per cent. of the families had been visited by three or more different workers in addition to the health visitor. Most of these families were of the " problem family " type, where multiplicity of visiting is a well-known phenomenon. Multiple visiting was more frequent in families

[1] *Med. Offr.* **91**, 83–5. ' A note on multiplicity of home visiting by medico-social workers ', R. C. Wofinden, M.D., D.P.H., Deputy Medical Officer of Health, Bristol.

in which there were more children in the household, greater degrees of overcrowding and poor social and evironmental standards, so the author concludes that most effort appears to be made on behalf of those most likely to be in need of assistance.

After reading this article we look forward, with interest, to the findings and recommendations of the Ministerial Working Parties, which are at present studying the function of the Health Visitor, the Home Nurse and other social workers.

News from Societies

COLLEGE OF GENERAL PRACTITIONERS SOUTH WEST OF ENGLAND FACULTY

In its first year the Board of the Faculty has concerned itself with establishing general practitioner research and with post-graduate study. Dr. Mallett, the member for research, has put in much time. A Faculty Advisory Panel has been formed, with Professor Milnes-Walker and Dr. A. H. Gale from Bristol University. After much preliminary work by Dr. Mallett of Wimborne, Dr. Hickson of Chippenham, and Dr. Cookson of Hucclecote, Glos., an Obstetric Survey of the Region was started on 1st June. This will run for a year. Dr. Mallett has also devised, with the help of Mr. John Dodd, Hon. Assistant Secretary, a system of quick warnings to members on the research register of any epidemics in or approaching the Region.

Post-graduate study has made a good start. From the founding of the faculty Dr. A. H. Gale, Director of Post-graduate Studies, University of Bristol, has given us every assistance. He is a co-opted member of the Faculty Board. With Dr. Jackson of Crediton he organized a most successful weekend course at Exeter. The recent week's residential course held in Bristol has been equally successful. It is hoped to hold regular weekend and week courses in future. The Faculty is represented on the Post-Graduate Advisory Committee.

Undergraduate Education. Dr. Hickson of Chippenham is the member of the Board in charge of this. Bristol University have already started a two-week training in General Practice for final year students, and two lectures in General Practice are given each year. The two-week course seemed to work very well. From the General Practitioners' point of view March and April are somewhat busy months, and for the students it is perhaps too near the Finals for them to give their undivided attention.

It is hoped that the membership will continue to increase. In the first year we have reached 221. It is a good start, but we would like to see many more join us.

D. M. CAMERON,
Hon. Sec.

BRISTOL MEDICO-CHIRURGICAL SOCIETY, 1953–54

The yearly work of this society has now acquired a pattern, almost a rhythm, for which the secretary at least is grateful. Annual General Meeting and Presidential Address are followed by a Clinical Meeting in November. The organizers of this do a great deal of hard work, and the staff of the hospital concerned co-operate nobly to produce an instructive yet delightfully informal evening. In recent years a custom has arisen of a discussion of selected cases following the demonstration of the patients.

The rest of the year's meetings are concerned with scientific papers and again by custom two of these are read by guests, one of whom is invited personally by the president. This session the A.G.M. and clinical meeting have already been reported in the *Journal*, and the presidential address is to be published soon. Mention has also been made of Mr. Cooke's paper in December.

Our two guests were Sir George McRobert (presidential) and Dr. G. F. Abercrombie (society). The former gave a masterly address at the February meeting on Tropical Medicine, while in April the second delighted us with a philosophical discourse on General Practice.

In January there was an excellent talk by Mr. FitzGibbon on plastic surgery—beautifully illustrated of course. In March Dr. Beryl Corner and Dr. Campbell combined forces to open a discussion on meningitis. Members were left secure in the knowledge that Bristol is the best place in which to suffer from this disease.

In May we had a stroke of good fortune. The visit to Britain of the American College of Surgeons brought several distinguished men, and thanks to the persuasive powers of Professor

Milnes Walker we were able to listen to Dr. Frederick Coller of Ann Arbor, Michigan, on early diagnosis in carcinoma of colon, a masterly talk by a master, simply preaching the simple virtues, and surprising to some from an American, praising bedside examination and history above all else.

BRIAN MCCONNELL, *Secretary.*

OFFICERS FOR SESSION 1954–55

President: Dr. Gordon Heron. *Treasurer:* Dr. J. MacRae. *Secretary:* Dr. B. E. McConnell.

PROGRAMME

(*Second Wednesday of the month at* 8.15 *p.m.*)

October 13th: General Meeting. **Presidental Address:** Dr. Gordon Heron.

November 10th: Clinical Meeting at Bristol General Hospital.

December 8th: **"Bladder Neck Obstruction":** Mr. Ashton Miller and Mr. J. P. Mitchell.

January 12th: **Report from America:** Dr. J. Cates, Dr. J. Cosh, Mr. J. Peacock and Mr. M. G. Wilson.

February 9th: Dr. McDonald Critchley.

CLINICAL SOCIETY OF BATH, 1953–54

Seven meetings were held during the session and the following papers were given:

In October 1953 Mr. J. Bastow discussed **"Arthroplasty of the Hip"** with a demonstration of cases. This was followed by a colour film prepared in Bath by Mr. A. E. Burton showing the stages in the cup arthroplasty.

In November, Dr. J. O'Neill read a paper on **"Generalized Lymphadenopathy"** and quoted a series of 100 cases which presented a picture somewhat similar to infectious mononucleosis. This paper was then discussed.

At the December meeting, Mr. B. Blair discussed the effects of **"Hexathide in Toxaemia of Pregnancy,"** with special reference to the pre-eclamptic state.

At the January meeting this year the Society welcomed Professor Milnes Walker who gave a most interesting talk on **"Cardiac Surgery"** which was illustrated by two films and very greatly appreciated.

At the February meeting the speaker, Mr. T. Tizzard, was seriously and unexpectedly indisposed on the morning of the meeting and, as a result, short papers were given on the following subjects:—

Dr. D. Pugh: **"Nephrotic Oedema treated by Acupuncture."**

Mr. P. Sames: **"Surgery of the Rectum."**

Dr. G. Kersley: **"The value of deep X-ray therapy in Arthritic Conditions."**

Dr. J. Bolton: **"Peptic Ulcer in the Aged."**

Mr. S. Glaser: **"The Preservation of the Hydronephrotic Kidney."**

In March the Society was addressed by Dr. John Hunt, the Secretary of the College of General Practitioners, on the Development and Aims of the College and present trends in general practice in this country. His paper was most warmly received and excited prolonged discussion.

At the seventh and last Meeting a round-table discussion on **"The Use and Abuse of Antibiotics"** was most usefully developed from the point of view of the General Practitioner, the General Surgeon, and the Pathologist.

THE OFFICERS OF THE SOCIETY 1954–55

President: Dr. J. R. O'Neill.

Future President: Mr. J. Bastow.

Committee: Dr. L. D. Brice; Mr. S. Glaser; Dr. G. D. Kersley.

Meetings will be held on the second Friday of every month from October until April and the October speaker will be Mr. J. M. Sanson who will discuss **"Some quoted Midwifery"**.

DEVON AND EXETER MEDICO-CHIRURGICAL SOCIETY, 1953–54

The Society had a successful year, its last meeting being in April. It has a membership of 194 and the average attendance at ordinary meetings is between 30 and 40.

There were four Clinical meetings preceded by quarter-hour talks on subjects of current interest such as "Some Assorted Skin Conditions"—Dr. John Simpson; "The Role of Fluoride in the Prevention of Dental Decay"—Mr. Jeffrey Fletcher; "Perirenal Pneumograms"—Mr.

Alan Gairdner; " Dysphagia Associated with Lung Complications "—Dr. C. J. Fuller. Two Clinico-pathological Demonstrations were held. There was a Symposium on " Cortisone and A.C.T.H." to which the contributors were Dr. C. J. Fuller, Dr. J. R. Simpson and Dr. J. O. P. Edgcumbe. Mr. Philip Scott gave a lecture on the " Fenestration Operation ".

Mr. Norman Capener gave his Presidential Address on March 18th and to it were invited the wives of members, the Dean of Exeter and Mrs. Wallace, and other distinguished persons. There were 140 people present and there was on view a representative display of drawings, hospital minutes, correspondence and portraits of members of the medical staff of the hospital in the eighteenth century.

Mr. Capener spoke on " The Hunterian Influence in Exeter ". He referred to the early provision made for the sick of the city up to the foundation of the hospital in 1741 and told the company something of such distinguished early members of the honorary medical staff as Dr. Thomas Glass, Dr. Patch, Dr. Blackhall and Dr. Lee Dicker and of the painter Opie who, like Turner and Reynolds, was a West Country man.

He traced the relationships as teacher and pupil of Cullen, William and John Hunter, Astley Cooper, Abernethy, Charles Bell and our own John Sheldon.

Mr. Capener showed on the screen a number of beautiful portraits including that of John Haddy James about whom there is much biographical material and who served as medical officer to the Guards Regiment at the battle of Waterloo. A relation, Miss Farringdon of Lympstone, was a guest at the meeting, thus completing our link with the past. Mr. Capener showed a number of treasures from the Cathedral Library including first editions of Harvey, Booerhaave and John Hunter, and others from the fine collection in the Hospital Medical Library.

OFFICERS FOR SESSION 1954–55

President: Mr. Norman Capener.
Treasurer: Dr. G. Stewart Smith.
Secretary: Dr. Charles Seward.

PROGRAMME

(*All meetings are held on Thursdays in the Library of the Royal Devon and Exeter Hospital unless otherwise stated.*)

October 28th, 3.30 p.m.: Mr. Osmond-Clarke, C.B.E., F.R.C.S.: **" Pain in the Neck and Arm."** (South Western Dinner.)

November 4th, 3 p.m. (Clinical Meeting): Dr. R. B. H. Tierney, M.D.: **" Differential Diagnosis of Jaundice."**

November 18th, 8.30 p.m.: Clinico-pathological Demonstration.

December 16th, 8.30 p.m.: Dr. F. S. W. Brimblecombe, M.D., M.R.C.P.: **" Some Common Disorders of Childhood."**

January 20th, 3 p.m. (Clinical Meeting): Mr. Philip Scott, F.R.C.S.: **" Diseases of the Ear, Nose and Throat "** (with film).

February 17th, 8.30 p.m.: Clinico-pathological Demonstration.

March 17th, 8.30 p.m.: Dr. J. O. P. Edgcumbe: **" Miscellaneous Haematology."**

April 14th, 8.30 p.m.: Dr. Ffrangçon Roberts, M.A., N.D., F.F.R. (Title awaited.)

April 28th, 8.30 p.m.: Clinical Meeting, at the Princess Elizabeth Orthopaedic Hospital.

PLYMOUTH MEDICAL SOCIETY (FOUNDED 1794) 1953–54 SESSION

Despite its antiquity the Society goes from strength to strength, and last season the numbers both of members and attendances at meetings exceeded all previous records. The Society still has the original minute books dating from its foundation in 1794. They show, among other things, the priority of discovery by Dr. E. L. Fox of the treatment of myxodema by thyroid gland in 1892. During the year the Library Committee of the B.M.A. cast covetous glances upon these books, but after a succinct debate the Society decided that they should remain in Plymouth, although loan for special purposes would be considered. The society also possesses two portraits of Edward Jenner; an original by Northcote was regarded by Jenner himself as the portrait which he liked best. It has recently been restored with the co-operation of the National Portrait Gallery. An excellent copy of this portrait was for many years owned by the Royal Albert Hospital, the Committee of which saved it from the cold hand of bureaucracy by presenting it to the Society in the early months of 1948. It continues therefore to adorn that part of the Hospital which so soon lost its royal title to become the Devonport " section ", a designation which must have been inspired by some bureaucratic microtomist.

Last year's President provided an innovation by inviting Professor A. L. Rowse to give a mid-sessional address. His title was " Elizabethan England and Today "; his theme was the outburst of inspired activity which permeated England in the first Elizabethan reign and his

belief that the present reign was about to witness a similar epoch in which discoveries of science, and among them those of medicine, would represent the discoveries of the Elizabethan adventurers. Speaking without a note, in a conversational style and with almost Gallic gestures, he carried us from the home life of a first Elizabethan lady through the achievements of the latter part of that reign to his vision of our not too distant future. It was a fascinating and inspiring address.

Perhaps it was hardly a coincidence that our next visiting speaker was Sir Russell Brain. It must be a rare, and probably an unique, event in the long life of our Society, to be addressed by the President of the Royal College of Physicians, and to hear an original exposition of the subject which, despite his many other activities, he had made his own. Cervical spondylosis became a reality in a way that no journal could make it.

Against this background other speakers might well feel that they flatulated against thunder. Far from it. Large and enthusiastic audiences heard Dr. Macdonald Critchley expose the intricacies of " The Body Image ", but it must be admitted that most of us, once separated from Dr. Critchley's persuasive diction, left this image to his academic care. Dr. P. M. F. Bishop's sound advice, backed by his researches, on " Endocrine Therapy " rapidly restored us to the fundamentals of practice, while our annual visit to the Royal Naval Hospital not only revealed the wealth of clinical problems encountered there but enabled us to hear at first hand of atomic explosions and of problems of high-altitude flying. In the face of all this talent our local boys made good. Great interest was aroused by Doctors Gilroy and Sweet on the " Psychological approach to Pruritus Ani ", by Dr. Jolly on " Hermaphrodites " and by Dr. Forbes on the " Treatment of Hemiplegia ". Three clinical evenings, rich and strange in their exhibits, completed the programme. Tigers as we are for work, our Annual dinner and dance was no less memorable than its predecessors. *Floreat aeternum.*

Dr. H. H. Cohen succeeds Mr. C. S. C. Prance as President for 1954–1955. Other officers are unchanged, with Mr. R. Howarth as Honorary Treasurer, Dr. C. R. Croft as Honorary Librarian, and Mr. W. B. Waterfall (3 Hartley Avenue, Plymouth) and Dr. J. W. N. Owen (Castlehayes, Plympton), as Honorary Secretaries.

SOUTH SOMERSET MEDICAL CLUB

The South Somerset Medical Club membership is now approximately 50 and meetings of the Club are at present held on the third Thursday of the odd months of the year.

At the Annual General Meeting last year, Dr. S. Brimblecombe, Stoke-under-Ham, was elected President; Mr. J. A. Vere Nicholl, Vice-President, and Dr. P. P. Fox, Hon. Treasurer and Secretary.

A Clinical Meeting was held in November when Dr. Gardiner Hill of St. Thomas's Hospital gave an address on the Adrenal Glands.

In January there was a successful dinner and dance at which members invited guests.

In March Professor Milnes Walker gave an extremely interesting and instructive talk on Recent Advances in Cardiac Surgery which was illustrated by coloured films, and in May Dr. A. M. Best of Yeovil gave a very interesting talk on Medical Experiences as a Japanese P.O.W. in Malaya and Burma.

Forthcoming events include the Annual General Meeting which has been arranged for September. On November 18th Sir Arthur Porritt has agreed to come and address the Club, and it is hoped to arrange a joint meeting with the local branch of the B.M.A. for this occasion, and in January a Dinner and Dance will be held.

TORQUAY AND DISTRICT MEDICAL SOCIETY

Session 1953–54

The Society's activities have continued on a slightly reduced scale. Nine new members were elected during the Session, and the total membership is now 124.

Speakers during the Session were:

Dr. Alice Stewart: **" The Day to Day Work of a University Department of Social Medicine."**

Sir Zachary Cope: **" The Birth of Modern Scientific Medicine."**

Dr. G. H. Tovey: **" Salt and Water in Clinical Medicine."**

Dr. Meredith Davies: **" Is the Health Visitor really necessary? "**

Dr. Margaret Jackson: **" Investigations and Treatment of Subfertile Marriages with special reference to the Post-Coital Test."**

Professor Bruce Perry: **" Rheumatic Heart Disease."**

Dr. F. E. Camps: **" Death at Rillington Place."**

Dr. Hugh Jolly: **" Common Paediatric Disorders in General Practice."**

The average attendance at 6 Ordinary Meetings was 36 (30 last year). A Clinical Discussion on Health Visitors was attended by 20 members; the usual Clinical Meeting in conjunction with the S.W. Branch of the B.M.A. was again well attended, 50 signing the book.

A Medico-Legal subject was chosen for a joint meeting with Lawyers, Coroners, Dental Surgeons and Police; 112 were known to have been present. Dr. F. E. Camps spoke on the Christie murders at Rillington Place.

The Dinner and Dance after the Opening Meeting was again held at the Palace Hotel, and 120 members and guests were present.

A most interesting Summer Meeting was held at the Manor House, Torquay, including an address by the Warden on Rehabilitation of the Blind.

The Officers for 1954–55 are:

President: Dr. R. L. Midgley.

Joint Hon. Secretaries: Dr. I. R. Sutherland and Dr. C. P. Warren.

Honorary Treasurer: as before.

Honorary Librarian: as before.

CORNWALL CLINICAL SOCIETY

The Annual Meeting of the Society was held at the Camborne—Redruth Hospital on Friday, 8th October, 1954, when the following Officers were elected for the coming year:

President: Mr. T. M. Banham, F.R.C.S., *Treasurer:* Dr. W. L. Stewart, *Secretaries:* Dr. J. D. Hardy, Dr. T. S. Wilson.

The retiring President, Dr. Pollock, gave an address on **"Glycosuria and Pregnancy"**.

B.M.A. BRISTOL DIVISION

Clinical Meeting at Frenchay Hospital, Bristol

A Clinical Meeting was held by the Bristol Division of the B.M.A. in conjunction with the Branch, at Frenchay Hospital, on Wednesday, 16th June at 8 p.m. One hundred and eighty members and friends were present. In the absence of the Branch President, Professor Neale as Chairman of the Bristol Division presided.

Surgical cases were shown by members of the staff of the departments of neuro, thoracic, orthopaedic and general surgery and Dr. Pearson showed cases of chest disease. Mr. Jones of the University Veterinary Department, gave a demonstration of comparative medicine and Dr. Gaskell a series of X-ray films and techniques. An unusual, but welcome departure for a clinical meeting was an exhibition, arranged by Mr. Fitzgibbon, of ways and means of preventing burns and scalds.

Later, Mr. Alexander, Mr. Phillips, Mr. Belsey, Dr. Pearson, Mr. Routledge, Mr. Fitzgibbon, Mr. Jones and Dr. Gaskell presented the various cases demonstrated and under the stimulating Chairmanship of Professor Neale an interesting discussion was held.

In conclusion the Chairman thanked members of the hospital staff and in particular Mr. Bodenham who had done so much to make the meeting a great success.

R. C. Wofinden.

Programme of Events 1954–55 Sessions

October 27th: Medical films, in Main Physics Lecture Theatre, Royal Fort.

November 24th: Dr. Harvey Flack, M.D., Editor of *Family Doctor:* "*Telling the Public about Medicine*" (B.M.A. Popular Lecture—members may invite a friend), in Main Physics Lecture Theatre, Royal Fort.

January 26th: Dr. Stewart Smith, M.A., M.D., Area Pathologist, Exeter and Mid-Devon Hospitals Management Committee, in Main Physics Lecture Theatre, Royal Fort.

Spring, 1955 (provisional): Social event—a sherry party to be arranged for members and friends.

May 18th (provisional): Annual General Meeting, in Main Physics Lecture Theatre, Royal Fort.

June 15th: Clinical Meeting at Southmead Hospital, Bristol.

June 24th, 4 p.m. (provisional): B.M.A. Reception for newly qualified doctors (Bristol University), Harden Hall, Royal Fort, Bristol.

THE GLOUCESTERSHIRE BRANCH OF THE B.M.A.

The 1953–54 programme was brought to a close when the President (Dr. Logan) had as his guest Mr. Wylie McKissock, M.S., F.R.C.S., who delivered a stimulating address on the modern methods of investigation and treatment of cerebral vascular accidents. Mr. McKissock demonstrated, largely by the use of angiograms, how cerebral vascular accidents can be more accurately diagnosed now than in the past. Quite apart from the localization of haemorrhage occurring from an intracranial aneurysm he showed that many cerebral haemorrhages could be accurately localized and subsequently evacuated at operation, thereby removing what constituted a space-occupying lesion, with greatly improved changes of satisfactory functional recovery.

In the subsequent discussion it was evident that neurosurgical centres would be liable to inundation by patients suffering from cerebral vascular accidents if careful discrimination was not observed in the selection of patients for investigation. But Mr. McKissock laid stress on the fact that the results of treatment by surgery for cases of intracranial haemorrhage were so much better than those of treatment by the older conservative methods that Regional Boards would eventually be forced to increase the number of neurosurgical beds to cater for this new demand.

Officers 1954–55

President: Dr. T. B. H. Haslett.
President Elect: Dr. H. S. K. Lowry.
Vice President: Dr. J. Greene (Senr.).
Secretary: Dr. E. W. Hyde.
Assistant Secretary: Dr. R. E. Bowers.

October 14th: President's Address (Dr. T. B. H. Haslett), **" Use and Abuse of Antibiotics ".**
November 11th: **Clinical Meeting,** Gloucester Royal Infirmary.
December 9th: **Discussion on Pulmonary Tuberculosis.** Openers—Dr. F. J. Knights, Dr. R. H. Ellis, Dr. J. B. Hayward.
January 13th: To be arranged.
February 10th: Mr. J. F. Bourdillon, **" Osteopathic Methods in Orthopaedics ".**
March 10th: **B.M.A. Lecture.**
April 14th: **Clinical Meeting,** Cheltenham General Hospital.
May 12th: Dr. R. E. Bowers, **" Disappointments in Dermatology ".**

WEST OF ENGLAND CHILD HEALTH GROUP

Programme 1954–55

September 14th, 5.30 p.m. (Tea provided 4.45–5.30 p.m.): Mr. T. T. Stamm, F.R.C.S., Orth. Surg., Guy's Hospital: **" Recent Aspects of Foot Health."** (This lecture is arranged in conjunction with Dr. Menzies Cormack, Divisional M.O.H., Gloucester County Council, and is part of the " Foot Health Week " programme for South Gloucestershire.) In the Main Physics Lecture Theatre, Royal Fort.

October 22nd, 5.30 p.m. R. C. Wofinden, M.D., D.P.H., Deputy M.O.H., Bristol: **" Health and Welfare Services in Sweden ":** In the Children's Hospital.

November 11th, 7.45 p.m.: Members of the staff of the Children's Hospital. (N.B.—Cases on view 7.45–8.30 p.m. Discussion 8.30 onwards): **Clinical Demonstration on " The child who fails to thrive."** In the Children's Hospital.

December (date to be announced later), 8.30 p.m.: W. S. Craig, M.D., F.R.C.P.(E), F.R.S.(E), Professor of Child Health, Leeds University: **Subject to be announced later.** In the Main Physics Lecture Theatre, Royal Fort.

January 21st, 8.30 p.m.: Dr. Mary Sheridan, M.A., M.B., CH.B., D.C.H., Deputy M.O., Children's Department, Home Office: **" Speech and the Deaf Child "** (illustrated). In the Small Physics Lecture Theatre, Royal Fort.

February 25th, 5.30 p.m.: Mr. Vaughan-Davies, Music Adviser, Bristol Education Department: **" Music and the Child."** In the Children's Hospital.

March 25th, 5.30 p.m. Dr. S. C. Walker, M.D., D.P.H., Senior M.O., M. and C.W., Bristol; Dr. S. Hinds, M.D., M.R.C.P., D.P.H., Lecturer in Child Health and Social Medicine; Miss E. Duncan, B.A., B.SC., Statistician, Health Dept.; Miss M. Reed, Welfare Officer, Health Department: **" Unmarried Parents."** In the Children's Hospital.

April 29th, 8.30 p.m.: Dr. Grace Woods, M.B., B.S., D.P.H., D.C.H., Department of Child Health; Dr. H. M. Gibb, M.B., CH.B., D.P.H., Health Department; R. V. Saunders, M.A., B.ED., Educational Psychologist, Bristol: **" The Assessment of Cerebral Palsy "** (illustrated). In the Children's Hospital.

Notes and News

Mr. R. V. Cooke has been appointed to a Hunterian Professorship of the Royal College of Surgeons. His subject will be "Advanced Carcinoma of the Colon".

Surgeon Commander J. L. S. Coulter, D.S.C., edited the Naval Medical Services volume of the Official Medical History of the War. He has been awarded the Gilbert Blane Medal.

The following have left or are leaving shortly to take up consultant posts elsewhere:
Dr. G. M. Colson, Senior registrar (medicine) to Bolton.
Dr. B. M. Laurence, Senior registrar (paediatrics) to Derby.
Mr. P. S. London, Senior registrar (orthopaedics) to Birmingham.
Mr. H. F. T. MacFetridge, Senior registrar (ophthalmology) to Herefordshire.
Mr. P. Ramsay, M.B.E., Senior registrar (surgery) to Windsor.
Mr. T. F. Redman, Lecturer (Obstetrics & Gynaecology) to Leeds.
Dr. I. M'D. G. Stewart, Senior registrar (medicine) to Blackpool.

Birthday Honours: R. F. Bolt, O.B.E.
M.D. (Bristol): N. C. Tanner.
F.R.C.S. (England): W. J. Gall.
M.R.C.P. (London): June K. Lloyd, C. D. R. Pengelly, R. H. R. White.

UNIVERSITY OF BRISTOL

M.B., Ch.B. (BRISTOL) (JUNE, 1954)

WITH FIRST-CLASS HONOURS

Mary E. Moore, (Distinction in Child Health, in Surgery, and in Obstetrics).

WITH SECOND-CLASS HONOURS

K. R. Gough (Distinction in Public Health and in Obstetrics); D. B. Paintin (Distinction in Obstetrics).

PASS

G. A. J. Ayliffe.
J. F. Baden-Daintree (Distinction in Public Health).
V. Baker.
C. A. Bickford.
J. M. B. Burn.
J. T. Bush.
T. H. P. Chatfield.
J. R. Clamp (Distinction in Public Health).
Mavis E. Coutts.
G. G. Crabbe.
Violet E. Dadds.
R. N. Davies.
G. N. Febry.
D. W. Fortune.
Angela C. Fowler.
D. B. Fowler.
J. S. Hughes-Games.
J. L. James (Distinction in Child Health).
F. E. S. Keiller.
Jean D. Kelly.
C. A. Milner.
E. J. R. Morgan.
D. G. Osmond.
J. M. V. Packer.
Ruth D. Penn.
M. Roylance.
R. K. M. Sanders.
H. Savery.
R. Seymour-Shove (Distinction in Public Health).
F. G. Spear.
D. M. M. Thompson.
D. H. Waldron.
M. R. Wills (Distinction in Public Health).
G. C. B. Winter.

MEDICAL POSTGRADUATE DEPARTMENT PROGRAMME

October 24th, 10.30 a.m., Department of Medicine, Royal Infirmary: **"Cortisone and A.C.T.H."** Dr. J. E. Cates.

October 31st, 10.30 a.m., X-ray Department, Southmead: **"Pulmonary Tuberculosis."** Dr. A. T. M. Roberts.

November 7th, 10.30 a.m., X-ray Department, Southmead: **"Pulmonary Tuberculosis."** Dr. A. T. M. Roberts.

November 13th } Course in Civil Defence arranged by Regional Hospital Board.
November 14th }

November 21st, at 10.30 a.m. and November 28th, at 10.30 a.m.: Dr. R. F. Barbour and the staff of the Child Guidance Clinic will give two demonstrations at the Child Guidance Clinic 7 Brunswick Square, Bristol 2.

Appointments

BRISTOL

Name	*Appointment*	*Formerly*
GORDON, IAN RONALD SIMSON, M.D., M.R.C.P., D.M.R.D.	Consultant Radiologist	Locum Tenens Assistant Radiologist, United Bristol Hospitals) S.H.M.O. Grade)
LAMB, W. R., B.A., M.B., B.CH. (DUBLIN), F.R.C.S.I.	Anaesthetic Registrar, Southmead Hospital, Bristol.	Senior House Officer in Anaesthetics, United Liverpool Hospitals.
TOVEY, L. A. D., M.B., CH.B. (BRISTOL).	Registrar to the South West Regional Blood Transfusion	Registrar in Pathology, County Laboratory, Dorchester.
FOX, J. H., M.B., CH.B. (SHEFFIELD), D.P.H. (EDIN.).	Registrar in Diseases of the Chest, Bristol Clinical Area.	Senior House Officer in Diseases of the Chest, Ham Green Hospital.
LAIDLAW, C. D'ARCY, M.R.C.S., L.R.C.P., F.R.C.S. (ENG.).	Surgical Registrar, Southmead Hospital, Bristol.	Surgical Registrar, St. Chad's Hospital, Birmingham.
DUTTON, J. E. M., M.B., B.S. (DURHAM), F.R.C.S. (ENG.).	Senior Registrar to the Dept. of Neurological Surgery, Frenchay Hospital, Bristol.	Registrar to the Dept. of Neurological Surgery, Frenchay Hospital.
LENNARD, M. B., M.B., CH.B. (BRISTOL), M.R.C.S., L.R.C.P.	Clinical Assistant in Orthopaedic & Traumatic Surgery, Weston-super-Mare General Hospital.	General practice.
CATTON, M. J., M.B., B.S. (LONDON).	Registrar in Pathology, Southmead Hospital, Bristol.	Graded Specialist in Pathology, R.A.F.
MILLS, C. P., M.B., B.S., F.R.C.S.	E.N.T. Registrar.	Surgical Registrar, Royal Hampshire County Hospital, Winchester.
CANTRELL, G. L. M.B., CH.B., D.O.	Senior Registrar in Ophthalmology.	Registrar in Ophthalmology, United Bristol Hospitals.
TULLOCH, L. G., M.B., CH.B., M.D.	Senior Registrar in Dermatology to Bristol Clinical Area based at U.B.H. (*R.H.B. appointment*)	Registrar, Manchester & Salford Hospital for Diseases of the Skin.
LLOYD, MISS J. K., M.B. CH.B.	Registrar in Paediatrics.	S.H.O. in General Medicine, United Bristol Hospitals.
BENNETT, J. R., B.A., M.B., B.CHIR., F.F.A.R.C.S.	Registrar in Anaesthetics.	Registrar in Anaesthetics, United Liverpool Hospitals.
FARRANT, P. C., M.B., CH.B., M.R.C.S., L.R.C.P.	Registrar in Pathology.	Demonstrator in Pathology, University of Bristol.
MORGAN, O. T. L., M.B., B.S., D.A.	Senior Registrar in Anaesthetics.	Registrar in Anaesthetics, United Bristol Hospitals.
ISAACSON, A. H., M.D., M.R.C.P.	Part-time Registrar in Radiodiagnosis in conjunction with University D.M.R.D. Course.	General Practice, Preston.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
RUDDELL, J. S., B.A., M.B., B.CH., B.A.O., D.A., M.A., M.D.	Consultant Anaesthetist, North Gloucestershire Clinical Area.	Consultant Anaesthetist, Northallerton Group of Hospitals.

NORTH GLOUCESTERSHIRE—continued.

Name	*Appointment*	*Formerly*
BALMFORTH, G. V., M.R.C.S., L.R.C.P., M.B., B.S., D.(OBST.), R.C.O.G.	Medical Registrar, Gloucestershire Royal Hospital, Gloucester.	Trainee Assistant in General Practice in Barnstaple.
MORGAN, RHONA E., M.B., B.CH. (WALES), D.(OBST.)R.C.O.G., D.C.H.	Medical Registrar, Cheltenham General Hospital.	Senior House Officer (General Medicine), Stoke Mandeville Hospital.
CUSACK, J. J., M.B., B.CH. (DUBLIN)	Clinical Assistant in Ophthalmology, Cheltenham General Hospital (4 sessions).	Surgeon Commander, Royal Navy—Ophthalmic Specialist.
JATOI, A. M., M.B., B.S. (BOMBAY).	Orthopaedic Registrar, Gloucestershire Royal Hospital, Gloucester.	Senior House Officer in Orthopaedics, Gloucestershire Royal Hospital.

BATH

Name	*Appointment*	*Formerly*
GERRARD, JEAN O., M.B., CH.B. (ST. ANDREWS')	Anaesthetic Registrar to the Bath Group of Hospitals.	Senior House Officer in Anaesthetics, St. Martin's Hospital, Bath.
POYNER-WALL, PHYLLIS, M.B., B.S. (LONDON), M.R.C.S., L.R.C.P., D.C.H.	Deputy Resident Physician, Winsley Chest Hospital, near Bath.	Registrar in Diseases of the Chest, Winsley Chest Hospital.
STEVENS, D. M., M.R.C.S., L.R.C.P., M.B., B.S., D.L.O. (ENG.), F.R.C.S. (EDIN.).	Consultant E.N.T. Surgeon to the Bath Clinical Area.	Senior Registrar in E.N.T. Surgery, United Cardiff Hospitals.
BAZELEY, R. W., M.R.C.S., L.R.C.P., M.B., B.S.	Re-appointed for a further year as Clinical Assistant in General Medicine, Royal United Hospital, Bath (1 session).	Is in general practice as Radstock.
PINCHING, J., M.R.C.S., L.R.C.P., B.M., B.CH., M.R.C.P.	Re-appointed for a further year as Clinical Assistant in General Medicine, Royal United Hospital, Bath (1 session).	Is in general practice at Wells.
IRWIN, T. W. N., M.B., B.CH. (WITWATERSRAND).	Surgical Registrar to the Bath Group of Hospitals.	Full-time study for the final F.R.C.S. (Eng.).
MATHESON, A. T., B.SC., M.B., CH.B. (CAPE TOWN), F.R.C.S. (EDIN.).	Surgical Registrar to the Bath Group of Hospitals.	Locum Surgical Registrar at the Central Middlesex Hospital.
RIVETT, P. A. H., M.B., CH.B. (LEEDS), M.D. (LEEDS), D.C.H.	Appointed for one year as Clinical Assistant in Paediatrics at the Royal United Hospital (1 session).	Is in general practice at Calne.
ARMIN, R. H., M.A., M.B., B.CHIR., M.R.C.S., L.R.C.P., D.P.M.	Consultant Psychiatrist and Deputy Physician Superintendent, Mendip Hospital, Wells.	Assistant Psychiatrist at Roundway Hospital, Devizes.

SOUTH SOMERSET

Name	*Appointment*	*Formerly*
SINTON, J. R., M.B., B.S. (LOND.), M.R.C.P. (LOND.), M.D. (LOND.).	Consultant Chest Physician to the South Somerset Clinical Area.	Deputy Physician-Superintendent & Resident Assistant Physician, Baguley Hospital, Wythenshawe, Manchester.

SOUTH SOMERSET—continued.

Name	*Appointment*	*Formerly*
HUGGILL, P. H., B.A., M.B., B.CH., M.R.C.S., L.R.C.P., D.L.O., F.R.C.S. (ENG.).	Consultant E.N.T. Surgeon to the South Somerset Clinical Area.	Part-time Consultant E.N.T. Surgeon to the Camberwell Group of Hospitals, part-time Senior Registrar to the E.N.T. Department at the Royal Cancer Hospital & Hon. Clinical Assistant to the E.N.T. Department at St. Mary's Hospital.

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
DREW, D. W. A., L.D.S., R.C.S., M.R.C.S., L.R.C.P.	Anaesthetic Registrar to the Plymouth, South Devon & East Cornwall Hospital.	Anaesthetic Registrar at the United Cardiff Hospitals.
AZAM, M. A., M.B., B.S., (PUNJAB), F.R.C.S. (EDIN.).	Surgical Registrar at the Plymouth, South Devon & East Cornwall Hospital.	Senior House Officer at Shotley Bridge General Hospital.
PARKER, R. D. H., M.B., B.CH. (DUBLIN), F.R.C.S.I.	Surgical Registrar at the Plymouth, South Devon & East Cornwall Hospital.	Surgical Registrar at Worcester Royal Infirmary.
PASCALL, K. G., L.M.S.S.A., M.B., B.S., F.R.C.S. (EDIN.).	Senior Casualty Officer to the Plymouth, South Devon and East Cornwall Hospital Group.	Assistant Senior Administrative Medical Officer, H.Q. Staff of the South Western Regional Hospital Board.
PEARSON, A. F., M.B., CH.B. (MANCHESTER), D.(OBST.) R.C.O.G., M.R.C.O.G.	Clinical Assistant in Obstetrics & Gynaecology at Stratton Hospital, Bude, & Old Tree House Maternity Home, Launceston (1 session).	Is in general practice at Holsworthy.
WHEELDON, F. T., M.R.C.S., L.R.C.P., M.B., B.S. (LONDON), F.R.C.S. (ENG.).	Consultant Orthopaedic Surgeon, Plymouth Clinical Area.	Senior Registrar in Orthopaedic & Traumatic Surgery, St. George's Hospital, S.W.1.
GREENBURGH, H., M.R.C.S., L.R.C.P., M.B., B.S. (LOND.), M.R.C.P. (LOND.), M.D. (LOND.).	Consultant Pathologist, Plymouth Clinical Area.	Senior Registrar in Pathology, Southern Group Laboratory, Hither Green.
VARMA, R. M., M.B., B.S. (MADRAS).	Surgical Registrar at the Royal Cornwall Infirmary, Truro.	Senior House Officer to the Department of Neurological Surgery, Frenchay Hospital, Bristol.
RICH, W. M., M.R.C.S., L.R.C.P., D.O.M.S.	Clinical Assistant in Ophthalmology to the West Cornwall Clinical Area.	
BENNETT, H. S., M.B., CH.B. (ABERDEEN), D.M.R.D. (ENG.).	Consultant Radiologist to the West Cornwall Clinical Area.	Assistant Radiologist to the Department of Radiodiagnosis, Aberdeen General Hospitals.

Editorial

The last number of this *Journal* appeared in October 1954. Mr. H. Keith Lucas has bravely tackled the task of editorship for four years. But he has had less and less time for this and, regretfully, he has now resigned. The Medico-Chirurgical Society owes him much, for without his help this journal would have perished four years ago. And without its *Journal* the Society would have lost one of its proudest features. In its new form the Medical Journal of the South-West has served to bring together the medical societies and clubs scattered through the south-west region. Most of the credit for this improvement also is due to the vision and energy of Mr. Lucas.

The articles published in this *Journal* fall into two broad groups. There are those papers which have already been read before this Society or before other Societies in this region. There is plenty of this matter awaiting publication in future numbers. Secondly there are original papers by younger men—often registrars—who report cases or describe their own work. The supply of papers of this group is low, and contributions of this nature will be welcome.

COUSIN MARRIAGE

FORTY-SECOND LONG FOX MEMORIAL LECTURE DELIVERED ON 20TH OCTOBER, 1953

BY

J. A. FRASER ROBERTS, M.A., M.D., D.SC., F.R.C.P.

Director, Burden Mental Research Department, Stoke Park Colony, Bristol

It is a great honour to be invited to contribute a lecture to this long-established series, founded to commemorate a very eminent physician of the Bristol Medical School and a member of a family distinguished for services to medicine and to the City over many years. I have also seen with particular pleasure that the first Long Fox lecture was given by John Beddoe, a pioneer of British anthropology and one of the greatest men of science that this City has produced. His writings, particularly *The Races of Britain*, have been an inspiration; and, like many others, I have turned to his tables as a source of material. I have been grateful, too, to have had available at the University library the Beddoe collection of books, which contains some rare and useful works.

Turning to the subject of this lecture—cousin marriage—you will naturally expect a biologist to deal with the biological implications and consequences of the marriage of blood relatives; and this indeed is what I propose to do. But before starting on the main subject I should like to spend some of the time on more general questions; for in addition to biological implications there are others—for example, the sociological—and these other aspects of our subject are important and, I think, interesting.

There can be few if any stronger taboos than those prohibiting the marriage of certain relatives. The scope of the prohibited degrees has varied widely at different times and in different places, sometimes extending to relationships which seem remote to us. The closest relationships, however, for example brother and sister, or parent and child, have at almost all times and in almost all places been considered an absolute bar. The very few exceptions only serve to emphasize the universality of something deeply rooted in the mind of man from very early times. The exceptions in fact attract undue notoriety by their very rarity. The brother-sister marriages amongst some of the Pharaohs arose from dynastic considerations, for it was held that a wife of a royal line transmitted the right of kingship to her children, and princes had an unquestionable right to the throne only if they were born of a father and mother who themselves had equal rights. It has been said that during the times of the Ptolemys the practice spread through the population, but such instances only emphasize how fleeting have been the departures from what many would no doubt hold to be a part of natural law.

Is there any stronger taboo than that against incest? It is difficult to think of one. Are there any other laws so relatively easily broken and yet so universally respected? But how have the taboos come to be established? What purpose do they serve? There are two explanations, the genetic and the sociological, or rather, I should say, the psychological-sociological. The genetic explanation, which seems very unlikely, is that from very early times mankind became aware of evil consequences arising from inbreeding, both in his own species and in his domestic animals. The sociological explanation is that the prohibitions are calculated to preserve the stability of the family. In any society the family is the all-important unit, and nothing could be more destructive of its harmony than the possibility of unions between its members. This is what Malinowski* says: "The main sociological reason for marriage taboos and prohibited degrees is the elimination of sex from relations of the family type. A group

* For this and a number of other references, I am indebted to "Kindred and Affinity as Impediments to Marriage, being the report of a commission appointed by His Grace the Archbishop of Canterbury". London, S.P.C.K., 1940.

leading a joint life with the intimacy of daily concerns, with the need of an organized authority and unselfish devotion, cannot tolerate within its framework the possibilities of sexual approaches, for these act as a competitive and disruptive force incompatible with the even tenor and stability of the family."

It is easy to see how well the psychological-sociological explanation fits the facts and how badly the genetic. The extension of prohibitions from the universally agreed close relationships to those which are distant can be seen to follow closely the pattern of family life—of what at a particular time and in a particular place constitutes the family group. For example, in some matrilineal societies marriages to aunts or cousins on the mother's side are strictly forbidden, whereas genetically equivalent marriages to a cousin on the father's side or to the father's sister are freely allowed, and may indeed be considered positively desirable. Or, to take another example, first cousins, the children of brothers and sisters, have often been considered as the equivalent of brothers and sisters. When this has been so, marriage between first cousins has been as much barred as marriage between brothers and sisters.

Then again, the genetic explanation can only apply to blood-relationship and not at all to relationship by affinity, that is, relationship created by marriage. There can be no genetic reason why a man should not marry his son's wife, for they have no biological connexion, yet the prohibitions apply just as strongly to affinity as to blood-relationship.

If we turn to what is allowed and what is not allowed here and now, we see at once that it does not make genetic sense, though it does make sound sociological sense. In this country a man may not marry his niece; but to anticipate what I shall be talking about a little later, he can do what is exactly equivalent genetically, namely, marry his double first cousin (double first cousins arise when, say, two brothers marry two sisters). Of course, a man may not marry his sister, yet it is possible for him, genetically speaking, to do just that, and perfectly legally. Suppose a pair of identical twin brothers were to marry a pair of identical twin sisters—not a far-fetched possibility. Speaking genetically, the offspring of the two couples are the same as brothers and sisters. Yet there is no legal impediment to the son of one couple marrying the daughter of the other.

The idea that mankind, from primitive man onwards, observed that bad results came of inbreeding, either in his own species, or in his domesticated animals, so giving rise to the taboos, is somewhat fantastic. First of all, it is doubtful how far he would have observed it; he certainly could not have observed it as a constant phenomenon. Some of the Pharaohs who came of quite a long line of marriages of very close relatives, were persons of the highest qualities, both physically and mentally. Thus, at the beginning of the Eighteenth Dynasty, perhaps the most brilliant in the long history of Egypt, there was a succession of highly gifted, and also highly inbred, rulers. Queen Hatshepsut, outstanding even in that company, was the daughter of half brother and sister and her mother Aahmes was the result of two successive marriages of full brothers and sisters. Of course, I am not suggesting for a moment that such marriages might in any circumstances be a good thing even from the narrow genetic point of view; all I am doing is emphasizing how thin is the attempt to explain the origin of the prohibitions on genetic lines.

It is true that in ancient writings statements can be found to the effect that the offspring of unions of blood-relatives tend to turn out badly; but on the whole I imagine that such references are rather rare. Pope Gregory the Great in a letter to St. Augustine refers to heathen laws permitting marriages between brothers and sisters and first cousins. He says: "We have learned from experience that the children of such marriages do not do well." Benedict the Levite, writing in the year 842, says: "Such marriages are the cause of physical defects in the children"—an interesting observation. The impression one gets, however, is that the writers were very naturally finding additional reasons to support prohibitions already established on other grounds. And, of course, there is a reason, though not an intrinsic one, why in man poor results might sometimes be observed. People who defy such universally respected rules must

often be peculiar. They may transmit undesirable qualities to their offspring by way of inheritance, and are also likely to provide them with a poor home in which to grow up.

I am tempted here to digress a little and say a word about biological superstitions. There are those who say that ancient folklore and widely held popular beliefs are very likely always to have something in them, and that they should be treated with great respect. This was the view of an old friend of many years ago, who was a professor of chemistry. Now it is rather easy to be tolerant of superstitions in someone else's science; a professor of chemistry is unlikely to encounter a popular superstition to the effect that common salt is not composed of sodium and chlorine. At the present time the genetic theories of Lysenko seem to be most wholeheartedly supported by those least hampered by a knowledge of the subject. There are in fact biological superstitions which go back thousands of years and which are widespread even today, but which, nevertheless, are devoid of any foundation. A much more widespread popular belief than the evil consequences of inbreeding, which has something in it, is one which has nothing in it—namely, maternal impressions, the belief that the unborn child may be affected in some corresponding way by strong impressions made upon the mother. Of course, there is a case for investigating popular beliefs and Darwin investigated this one. He got a friend at a large maternity hospital to question some hundreds of women, before the birth of the child, as to strong impressions during their pregnancy. In not one single instance were any of these reflected in anything wrong with the child, or anything outstanding about the child. But, of course, after the child was born the mothers thought of all kinds of associations. Professor Darlington, in a recently published book*, gives an example showing how widespread this erroneous belief still is, even amongst educated people. Some years ago a lady wrote to the Tate Gallery, asking whether she could have a good look at Millais's well-known picture, " Sir Isumbras at the Ford ". At the time, for some reason, it was stored away. She was sure that if she could contemplate the picture her unborn son would look like the two delightful children, whom, you may remember, the artist also included. Well, biology is a funny science, and as things turned out, it wasn't a son—it was a daughter —and what she looked like was the horse. The much stronger belief has in fact led to little if anything in the way of rules of conduct; the lesser one, a belief in the evil effects of inbreeding, cannot be responsible for so important and mandatory a set of social rules.

So we are left with customs and laws which, we may agree, have a sound and useful sociological basis. The present laws of this country seem well adjusted to their purpose—the preservation of family life, without undue or unreasonable individual hardship. Only relatively close blood-relationship is a bar, or close affinity created by marriage. Of course, during a period of change there are liable to be difficulties. For some centuries during which the family unit was becoming smaller and progressively more restricted to closer relatives, the law tended to lag behind and the more widely drawn prohibitions of earlier times led to much difficulty. But it all seems to have settled down now. The Roman Catholic Church has attained the same end in a somewhat different way. They regard certain very close relationships as constituting a universal and unalterable bar. Others are dispensable. Even a brother-sister marriage is theoretically dispensable, but, say the canonists, no dispensation ever has been given for such a marriage, and none ever will be. Dispensations for the marriage of uncle and niece, in countries where such a marriage is legal, are much discouraged, are granted very rarely, and only for a good reason. Dispensations are freely given, I believe, for the marriage of first cousins.

There is little if anything left in the way of social problems, but there are biological consequences and implications and it is to these that I want to devote the rest of this lecture. As a preface I should like to say this: I should not like anyone to get the impression that I subscribe for a moment to the belief that we are wiser or cleverer than our predecessors. There is nothing more ridiculous than the dwarf on the giant's shoulder saying " Look how much taller I am than papa ". Rather, what most of us,

* *The Facts of Life.* London, Allen and Unwin. 1953.

I am sure, often feel is: "How I wish Newton, or Harvey, or Galton, had known about this; I wonder what he would have made of it." Science progresses by building up on foundations already laid; but, to change the metaphor, there come moments of illumination, and then nothing can ever be the same again. A key is discovered and a door is opened to a whole new world. In biology such a transcendent moment came in 1900 with the rediscovery of the work of Gregor Mendel. To quote Professor Darlington once again: " Before the year 1900, we may say, almost all that we believed of the principles of heredity that was not in Lucretius was wrong." The one exception was Mendel, and he was before his time. Darwin was not; in fact in regard to heredity he saw no further than the rest. So it is in the light of the transformation of 1900 and what has been learned since that we must assess the biological implications of the marriage of blood-relatives.

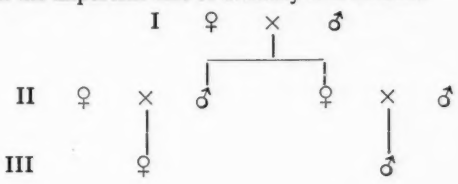
May I remind you of a very few basic principles of genetics which must underlie our discussion. The body is made up of cells. Each cell contains a nucleus, which is its directing mechanism. Within each human nucleus lie twenty-four pairs of chromosomes, all of characteristic size and shape. Along the length of the chromosome filaments are arranged the genes. They are in linear order: as is often said, like beads on a string. The total number is very large—many thousands, or indeed, more probably, many tens of thousands. The genes are the hereditary determinants. Everything that the parent contributes to the child by way of organic inheritance is contained in the gametes, the ovum and the spermatozoon, which are single cells; and the whole of that contribution is represented by the complement of genes. So if we follow the very simple essentials of chromosome behaviour, we have the key to understanding heredity.

When, during development, an ordinary cell division takes place, the forty-eight chromosomes, and the genes they contain, have doubled. Each filament has laid down alongside itself an exact copy. When the mother cell divides, giving two daughter cells, the doubled filaments separate, and so the daughter cells contain in their nuclei the very same chromosomes and borne upon their length the very same complement of genes.

Prior to the formation of the gametes, however, a division of a special type, the reduction division, takes place. The consequence is that the members of each pair of chromosomes separate bodily, so that an ovum or spermatozoon contains, not forty-eight chromosomes, but twenty-four, one derived from each pair. When fertilization takes place, each chromosome from the sperm nucleus pairs up with the corresponding chromosome from the nucleus of the ovum, and so there is reconstituted in the new individual the full somatic number of twenty-four pairs, one member of each pair having been contributed by the father and one by the mother. Starting with the all-important fact that the genes are paired, we see that if we think of one particular gene, on one particular chromosome of one particular pair, the chance that the mother—or the father—will contribute that particular gene to a particular child is one-half. We can extend this to all the genes, so parent and child have half their genes in common.

The same thing is true of brothers and sisters. Let us call the alternative genes *A* and *B*. The first child has a chance of one-half of getting *A* and one-half of getting *B*; so has the second child. It is like tossing two coins simultaneously. On the average, the result of every four throws is: two heads once; two tails once; and a head and a tail twice. So brothers and sisters, that is sibs, also share half their genes in common. By extending this reasoning, we find that grandparent and grandchildren have one-quarter of their genes in common. So have uncle or aunt and nephew or niece. So have double first cousins.

Let us look at the important case of ordinary first cousins.



If one of the cousins, III 1, possesses a particular gene, the chance that she received it from her father, II 2, is 1/2. If he carries it, the chance that his sister, II 3, also carries it is 1/2. If she carries it, the chance that she transmitted it to her son III 2, is 1/2. $1/2 \times 1/2 \times 1/2 = 1/8$; hence, if a person bears a particular gene, the chance of encountering it in a cousin is 1/8. We can generalize this: first cousins have one-eighth of their genes in common.

As blood-relationship gets more remote, the proportion of genes in common falls off rapidly. Second cousins, that is, the offspring of two first cousins, have one-thirty-second of their genes in common; third cousins only one in one hundred and twenty-eight. The whole biological significance of consanguineous marriages lies in these figures and you can see how much more important are the close relationships and how relatively unimportant those which are more distant. There is another point, too; the essential thing is the degree of relationship of husband and wife. Previous consanguineous marriages in the lineage of either are irrelevant.

Human beings differ in their hereditary qualities because their genes differ and this applies both to normal variations and to abnormal characteristics. From the point of view of assessing the consequences of cousin marriage, however, it is the definitely abnormal genes which are important. Genes are stable things in the ordinary way, rather like atoms used to be in chemistry, but every now and then a physico-chemical change takes place and the gene changes its character. If this should happen in the germinal tract, the new gene, which has arisen by this process of mutation, is thereafter transmitted just like the old one. Confining our attention to abnormal genes we see that there are two possibilities. First, the organism may require the two normal unmutated genes for its normal development. If, as the result of a mutation in a parent, it receives the abnormal gene, together with the corresponding normal gene from the other parent, the damage is done and the individual is abnormal. The good gene cannot overcome the effect of the bad one. Should the effect of the bad gene be so serious that the abnormal individual cannot live or grow up, that is the end. It cannot be transmitted further. If, however, the effect is less serious we see the direct transmission from generation to generation of something like polydactyly or congenital cataract.

But the transmission of these dominant abnormalities is irrelevant to our subject. Effectively, it is inheritance from one parent only, and so it does not matter whether they are related or not. It is the other possibility, the recessive defect, which concerns us. It often happens that there is a sort of reserve of normality. Only one of the genes making up the pair is required for normal development. One gene can, in fact, do the work of two. The person who has one normal gene and one abnormal gene is outwardly perfectly normal. Only when two people happen to marry, both carrying the same abnormal gene, can the defect appear. Albinism is due to a recessive gene. The carriers, with one gene for albinism, are outwardly normal, but, of course, the gametes are of two kinds, produced in equal numbers. When two carriers happen to marry, these are the possibilities: first, the union of two gametes both with the normal gene, giving a perfectly normal child; second, a normal gamete, with an abnormal; or third, the other way round, abnormal with normal. Both these offspring are carriers. Finally both gametes may happen to carry the abnormal gene, and so we get the abnormal child, in this case the albino. The result, then, on the average, in terms of what we see, is the familiar Mendelian ratio of three to one—three normals to one affected. Thus, recessive defects are familial in their incidence. Father and mother are always, or nearly always, outwardly normal, but more than one child in the sibship is frequently affected.

Recessive defects are all rare, or very rare, for a reason I shall mention in a moment. There are, however, a great many of them and so they are not negligible in the aggregate. But though the defects appear rarely, because carriers do not often happen to marry, the genes themselves are not so very rare. This is because carriers are so much more numerous than the affected offspring themselves, that is, individuals with two abnormal genes. I think that it is easy to see this, without giving a formal proof. Actually, if one person in 10,000 is affected, one person in 50 in the population must

carry the gene; if one person in 100,000 is affected, one person in 160 is a carrier; if one person in a million is affected, one person in 500 is a carrier. Now supposing a person is a carrier of one particular harmful recessive gene, with a frequency of carriers of one in 50, the chance that if he marries some unrelated person, she also will be a carrier is one in 50; but if he marries a first cousin, the chance is appreciably greater; actually, it is one in eight, as we saw a moment ago. In other words, whereas one marriage in 2,500 between unrelated persons is capable of yielding affected offspring, one marriage in 400 between first cousins is capable of doing so. If the abnormality is much rarer, say one in a million, one marriage in a quarter of a million between unrelated persons is capable of yielding affected offspring, compared to one marriage in 4,000 between first cousins.

It is indeed found that with rare recessive defects there is always an excess of consanguineous marriages between the parents as compared with the rate of cousin marriages in the population generally. If the defect is really very rare, say alkaptonuria, with an incidence of about one in a million, we expect and find that no less than about 40 per cent of all affected people are the offspring of first-cousin marriages.

So we see that an increased chance of the unexpected appearance of some recessive defect or other—and many of them are serious and unpleasant—is an inescapable consequence of the marriage of persons related by blood. What matters to individuals, however, is not by how much a small risk is relatively increased, but how great may be the total absolute risk. In fact, the increased absolute risk is not a very big one. The chance that any random pregnancy will end with some severe congenital abnormality or other, or that some serious developmental abnormality will manifest itself in early life, is about one in forty, and the added risk of a recessive defect turning up amongst the offspring of first cousins is probably less than this. Provided, of course, that there is no history of a recessive defect having already appeared in the family, the increase in the risk over and above the inevitable risks run by any couple who have a child is not of such an order that it need deter sensible and informed people and need not outweigh other considerations favourable to the marriage. We have a rather useful standard of comparison. In this country no fewer than one marriage in every eight is a marriage between a Rhesus negative woman and a Rhesus positive man. The consequent risk of haemolytic disease in second and later children is much greater than the risk of an undesirable recessive in a marriage of first cousins.

So we may conclude that the increased risk, though real, is relatively small. But though it may be ignored by individuals, even sensible and informed individuals, what about the consequences to the community? Let us think for a moment in an entirely theoretical sort of way whether it might be a good thing for the community if marriages of first cousins were prohibited. Of course, it is perfectly true that if marriages of first cousins were prohibited the total amount of recessive abnormality of all kinds in the population would be reduced to a certain extent. But we have to remember something else. Natural mutation is going on all the time, at the rates characteristic for each gene. Harmful genes are continuously being fed into the population, this being balanced by loss of genes, owing to the reduced fertility of their possessors. There is an equilibrium in these things. Incidentally, here is the reason why simply inherited defects are rare: it is because mutation is rare. The result of cutting down to some extent the proportion of affected children, those with two abnormal genes, would mean instead a corresponding increase in the number of carriers. Finally, after a long time, a new equilibrium would be reached. The original rate of manifestation would finally be restored; but there would now be many more carriers. A return to smaller populations and smaller breeding units would mean quite an outbreak of recessive defects of all kinds. So it does not seem that there is any strong case for interfering with the small amounts of inbreeding permitted and practised—that is important, practised rather than permitted—in our community.

This leads to a point which I might have mentioned before, but have postponed. How much consanguineous marriage is there in our population and is that amount changing? It certainly is. A hundred years ago, the proportion of first-cousin marriages

was perhaps one per cent, 10 per 1,000. In a large survey carried out on hospital patients in the middle thirties the rate was 6 per 1,000. There is some evidence, though the numbers are small, that it has fallen further, even in the short time that has since elapsed, and may now be no more than 4 or 5 marriages in every 1,000. The reasons are fairly obvious. Growth in population, urbanization, and increased mobility with recent developments of transport, all mean that the average person meets and has the chance of marrying an ever-widening selection of members of the opposite sex, and so is less likely to choose someone related by blood. The amount of recessive abnormality of all kinds must, in fact, be less than it was last century.

Now you may well ask: "How does all this fit in with the experience of the animal breeder? Doesn't he practice inbreeding and line-breeding, often of the closest kind, and on a vast scale? Hasn't it been an essential and very important part of breed improvement? Is there no possible application, talking quite theoretically, to the human species?" All this is perfectly true. Inbreeding is essential if fixity of type is to be attained. The extent to which it has been carried out in domesticated animals is in fact amazing. If we were to take at random any two Shorthorn cattle from any part of the world, the resemblance between them, talking in averages, would be greater than the average resemblance between father and son in that breed 150 or 200 years ago. In some domestic breeds line-breeding has been practised to such an extent that more than a quarter of all the genes in the breed are derived from one individual, that is, an individual who is effectively the grandfather of the whole breed. There are even breeds in which more than half the genes come from one individual, that is, genetically speaking, he is the father of the entire breed. The animal breeder secures fixity of the type he desires in this way; but in the process many individuals have to be sacrificed, partly in exposing and eliminating undesirable recessives. But he does not mind. In many circumstances it is well worth sacrificing fifty failures in order to secure one outstanding success. This could hardly in any conceivable circumstances apply to man.

But there is something even more important. The breeder knows what he wants. beef from the ox, milk from the dairy cow, speed from the racehorse or greyhound, eggs from the hen. Usually he wants one essential thing from each. Even when he only wants two he often finds himself in considerable difficulty. The successful dual-purpose animal is notoriously difficult to achieve: dual-purpose cattle, for example, bred for meat and milk simultaneously; or dual-purpose sheep, bred for mutton and wool. In man we do not want one thing, or two things, but hundreds of things.

What man has done to his domesticated animals is in fact remarkable: the floods of milk poured out by the dairy cow, the hundreds of eggs laid by the hen, the obesity of the prize ox. Even worse, the coat of the sheep bred for wool, a coat which renders it susceptible to the attacks of the blowfly, whose larvae eat it alive; a coat that would entangle it in every thorn bush, and which, if not removed periodically by the hand of man, would quite literally strangle its unfortunate owner. Worst of all, perhaps, are the ornamental breeds, bred for what must have been regarded as quaint and appealing oddities, but which are in fact pathological monstrosities; the Bulldog or the Pekinese, for example. What would happen if there were a return to primitive conditions if the protection of man were removed? Surely all these freaks would join in oblivion the dinosaur, the mastodon and the sabre-toothed tiger. In contrast to this, perhaps the most valuable characteristic of our species is its variability, so that different people can specialize successfully in hundreds of different tasks. Then there is the other all-important aspect of variability—the ability of the species to adapt itself to changing conditions. Flexibility is essential to survival, and the only way to secure and retain it is outbreeding or something near it.

The biological implications of cousin marriage were considered in terms of harmful recessive genes; and indeed this is the most important thing from the practical point of view. There is no time left to consider hereditary variations within the range of normality, so I will just say this. There are, of course, all kinds of interesting theoretical points, and some not so theoretical. But for the individual couple contemplating a cousin marriage there is little or nothing to worry about except the harmful recessive

genes. From the point of view of the community, there would indeed be problems, and of an interesting kind, if a very high proportion of the population married their cousins. But as we have seen, this is not so. Outbreeding is the rule and the relatively very small amounts of inbreeding actually practised do not have any more than a small effect on the distribution of normal, usually continuously distributed, characteristics, which are partly determined by heredity, and which distinguish one normal person from another.

I have come to the end of this brief review of cousin marriage and you may feel that I did not choose a very important subject; certainly not a controversial one. There is no burning problem here. But may there not be some kind of a lesson in that very fact? It is, after all, something that is related to very deep personal feelings. There has been trouble at times, but there is little or none now. We can consider biological implications in the calmest and most detached way. There is nothing here to produce that catch of the breath and rise of blood-pressure on opening the morning paper. Perhaps in some distant future—though there is little sign of it now—many other problems of life and conduct will fall into that happy state where there is an interesting past, but, to quote 1066 *and All That*, " history has now stopped ".

MENINGITIS IN CHILDHOOD

(Purulent and Tuberculous Infections)

BY

BERYL D. CORNER, M.D., F.R.C.P.

Consultant Paediatrician, United Bristol Hospitals and Southmead Hospital

MENINGITIS

" The man who starts for the first time to study the diseases of children is like a traveller in a foreign country; he hears a strange language spoken which he does not understand. At all events, if the language is not absolutely strange, it is spoken with a foreign accent, for the physical signs of disease are often different in children from what they are in grown-up people, so that you have to devote special attention to their interpretation." These remarks were attributed to Dr. Charles West, founder of the Hospital for Sick Children, Great Ormond Street, by Hutchison (1944).

Acute suppurative meningitis and tuberculous meningitis are most commonly encountered in childhood so that it is proposed to consider the clinical features of these conditions in children. Data have been obtained by analysis of cases admitted to the paediatric departments of United Bristol Hospitals and Southmead Hospital over the five-year period, 1948–53.

PURULENT MENINGITIS

A study of the literature reveals a good deal of inadequacy in description of the clinical features of this disease in its early stages and as it appears in different age groups in children. Henoch (1899), in his beautiful account of purulent meningitis which is well illustrated with case histories, describes the typical picture in the well-d veloped case as we see it in the child of school age and also mentions the high incidence of convulsions in infancy and the difficulty of diagnosis in the more chronic type of meningococcal meningitis. Reference to modern standard text-books shows a remarkable uniformity in the clinical description, " The onset of the disease is generally sudden and is characterized by vomiting, headache, sometimes pain in the back, temperature 102°–104°. There may be convulsions at the onset. From the beginning the signs of meningitis are generally evident ", etc. (Sheldon). This is a description of meningococcal meningitis which is familiar in the adult and in the older ages of childhood, but in the earliest years the language may be strange, the accent different. Hence, only too frequently is still seen the tragedy of the young child, who is sent into hospital so late in the disease, that the most effective drugs available are useless against the brain damage which is irreversible, and will either cause death or the survival of a useless individual.

In the past, a policy of procrastination before taking action in undiagnosed acute illness in early life gave little cause for concern as treatment was rarely effectual. Today treatment of the early case of acute purulent meningitis is so dramatic and rewarding with the antibiotic drugs that it is imperative that if a suspicion of this diagnosis crosses the doctor's mind immediate steps should be taken to get this confirmed by lumbar puncture, so that treatment may be started at the earliest possible moment.

In attempting to elucidate the problems of early clinical diagnosis, the records of 105 patients have been studied with particular reference to the history before admission to hospital and the important physical features on examination. These patients fell into four age groups, the disease in each of which seemed to present certain characteristic features which differed from those at other ages. Tables 1 and 2 and 3 show the

number of patients in each group, the mortality rate, and the type of organism causing the infection.

The high incidence of the disease (57 per cent of all cases) and the greater mortality rate in children under one year is at once apparent, but it should not be assumed that this age distribution is necessarily that always found in the general population, as owing to the special facilities for infant care at the hospitals concerned in this survey, a high proportion of young children with undiagnosed acute illness tends to be admitted to them.

The organism most frequently concerned is seen to be the *meningococcus*, but the low mortality rate with modern treatment in early cases is particularly notable. The only death occurred in a six-months-old infant who had been ill for twenty-four hours, and on admission was moribund due to fulminating meningococcal septicaemia which had caused suprarenal haemorrhage, the Waterhouse-Friderichsen syndrome.

Haemophilus influenzae commonly causes meningeal infection in infancy and in this series there were 2 deaths in 26 patients, which compared favourably with Alexander's (1947) result of 3 deaths in 25 patients. Pneumococcal meningitis carried the highest mortality rate which paralleled the findings of other authors, but in this series it seemed that these cases were particularly liable to be labelled "pneumonia" until the child became unconscious and beyond all hope of successful treatment.

There was a small number of cases in whom the cerebrospinal fluid showed changes typical of acute purulent meningitis but no organisms could be cultured. Some of these cases occurred early in the series when bacteriological methods were perhaps not so efficient as now and, in others, antibiotics had been given before admission.

It should be remembered that the symptoms and signs of purulent meningitis are produced by: (1) Bacterial toxaemia, (2) Cerebral irritation due to the presence of inflamed meninges and increased pressure of purulent cerebrospinal fluid and (3) Cerebral compression with cerebral oedema and sometimes vascular lesions in the brain. The dominance of any one of these factors gives us the varying clinical picture as seen in individual cases.

The clinical features in four age groups will now be considered. It is not proposed to ascribe clinical differences to the various bacteriological types of purulent meningitis, as apart from the haemorrhagic tendency in meningococcal infection, there were no very clear-cut distinguishing features in the cases analysed.

Neonatal Meningitis (*Table* 4)

There were fourteen children who developed purulent meningitis in the first few weeks of life. As in many infections of the newborn infant, the causative organism in seven cases was *B. coli*; the meningeal infection being the terminal phase of a septicaemia which arises from a local lesion in the renal tract, liver or lungs. This is a particularly fatal type of meningitis since, until chloramphenicol became available, treatment was usually unsuccessful. The only surviving case in this series was treated with that drug.

Nine children were less than ten days old, and in five of these the infection was probably directly related to problems of labour and delivery. In two cases, early rupture of the membranes followed by infection of the liquor amnii, produced antenatal infection of the infant, which might have been avoided by the administration of suitable antibiotics to the mother, when her membranes had been ruptured for twenty-four hours without the onset of labour. One infant, who died at the age of 21 days from *B. coli* meningitis, developed septicaemia three days after an abrupt change from full breast feeding to full cream dried milk. The disturbance caused by this sudden change in the bacterial flora of the upper intestinal tract may well have been responsible. In addition to the cases analysed, there were several infants with serious congenital abnormality of the central nervous system, e.g. meningo-myelocele, who developed terminal meningitis. These have been deliberately omitted from this analysis.

The pattern of symptoms in this age group is apparent from the table. The usual features are a change in colour of the skin, so that the child looks pale with a tendency

to episodes of cyanosis, fretfulness, anorexia and irritability. Unexplained fever may be noticed, or occasionally the temperature is subnormal. Examination revealed an infant whose greyish pallor was usually striking, but in whom no other physical signs could be found, although occasionally abnormal jerking of the limbs or rigidity were seen. In only five patients was abnormality of the anterior fontanelle evident, and neck rigidity was absent in all but three. Kernig's sign was invariably absent.

In the neonatal period the possibility of meningitis, or some other intracranial catastrophe, may be so easily overlooked that lumbar puncture is now one of the most commonly performed diagnostic procedures, and should never be omitted, or delayed if no other satisfactory diagnosis can be made to account for the child's symptoms. The only four children who survived in this series were diagnosed very early in the disease.

Infancy. One month to one year (*Table* 5)

At this age cerebral irritation was the predominant feature as a rule. Refusal to feed preceded all other symptoms and was often followed by irritability. A common history was that the baby slept fitfully, awakened frequently with a miserable but feeble cry, and in the words of one mother " whimpered like a puppy ". The high-pitched shriek often considered characteristic of meningitis was rarely noticed.

A very characteristic symptom commented on by nearly all parents was the child's dislike of being touched or nursed. This point requires particular emphasis as most sick children are comforted in their mother's arms, but the baby with early meningitis prefers to be left alone. Vomiting may be an early symptom but is not infrequently absent throughout the whole of the disease and in this series only twenty-two of the forty babies vomited before admission. Diarrhoea and signs suggestive of abdominal pain were occasionally present, when the child had attacks in which it screamed and drew its legs up; close observation sometimes revealed that this attack was actually a convulsion. Photophobia may occur but was only noticed once in this series of cases.

The second type of clinical picture is characterized by sudden lethargy and limpness which is due either to cerebral compression, or the overwhelming bacterial infection that, in meningococcal meningitis particularly, may give rise to haemorrhages and acute suprarenal insufficiency. Most mothers commented on the striking pallor of the baby and a few noted alterations in the respiration, such as grunting, rapid or irregular breathing, which sometimes was so pronounced that a diagnosis of respiratory disease was made. If convulsions occur they are a helpful diagnostic symptom but are rarely the earliest evidence of meningitis and Ounsted (1951) showed that convulsions were of serious portent. One baby was found in coma before the mother was aware of any illness.

A history of upper respiratory infection within the preceeding three weeks was found in almost every case irrespective of the type of clinical onset or the causative organism.

Clinical Examination. The diagnosis can be made or suspected almost entirely by inspection of the child, who should be examined in a good light and preferably unclothed. The posture and response to handling by the mother should first be noted; the baby usually lies still and limp but is sometimes restless and irritable, even so these children are usually too ill to put up any resistance to physical examination which is a helpful point in differential diagnosis.

The head should always be palpated and if the anterior fontanelle is still open, increased tension may be noted. This is however a variable sign, the tension being normal in many of our cases. The face is usually characteristic being pale with an expression of pain. The eyes tend to have a " far-away " look or may move unnaturally, sometimes there is photophobia and rarely a squint. Peculiar sucking and twitching movements of the mouth may be noted, and respirations are often grunting and rapid, or sometimes irregular or periodic. The pulse rate is variable, and in the comatose patients may be slowed due to cerebral compression. Temperature also shows great variation, being occasionally at hyperpyrexial levels, or in the shocked fulminating cases,

sub-normal. Neck and generalized rigidity of the trunk and limbs is sometimes present but was absent in many of the patients of this series, particularly in the younger age groups, and Kernig's sign is more often absent than present in infancy. A careful search of the skin may reveal a few petechiae or purpuric patches, but even in cases of meningococcal infection less than half of the children had a rash at any time.

Table 6. shows the four most important features noted on admission of the patient to hospital. The frequency of the finding of skin pallor may help the doctor to consider meningitis as a possible cause of the baby's illness, rather than some of the common but less serious infective illnesses of infancy, which more often cause flushed cheeks. It will also be seen that there was no meningismus in almost one-third of the cases at this age, and that a quarter were admitted in convulsions. In view of the frequency of convulsions at some stage of the disease in young children, it should be the rule that a convulsion occurring for the first time in a child with fever is an indication for lumbar puncture.

Older Age Groups

The clinical text-book picture is seen with greater frequency as infancy is left behind. The " one- to three-year-old " group of children was still very liable to convulsions in the early stage of the illness, but only one child over the age of three years had a fit. As in infancy, most of the children appeared pale and shocked when admitted to hospital and three-fifths of them were in coma or stupor, which demonstrated the deadly rapidity with which these diseases overwhelm the child before its nature is suspected by either parents or doctors. Over three years of age, the classical triad of headache, vomiting and prostration was present in every patient. As a rule in this group diagnosis presented little real difficulty.

Cause of Death (*Table* 7)

It is profitable to examine the reasons for the deaths in this series. Omitting the special group of neonatal cases, it will be seen that all these patients with one exception were infants. The salient features are the long history of symptoms before admission in six cases, all of whom had been labelled " influenza "; in two cases the child had been ill for fourteen days. These six cases were admitted in deep coma, with signs of shock. Eight deaths occurred within a few hours of admission. Two grossly abnormal children developed terminal meningitis. Lumbar puncture was performed in every case within a few minutes of admission and except in cases 11 and 12, intensive treatment was begun as soon as the purulent cerebrospinal fluid was seen. The two cases who died later than twenty-four hours after admission remained in coma with convulsions throughout that period, and showed little response to treatment although various antibiotics were tried according to the drug sensitivity of the organism. These cases serve to underline the tragedy of late diagnosis in this disease.

TUBERCULOUS MENINGITIS (TABLE 8)

The material comprised 84 cases of whom 68 were treated (cases still under treatment have been excluded). The remainder were considered too advanced to benefit at the time that streptomycin was in short supply. The survival rate was 65 per cent for treated cases. The classification of cases is that used by the Medical Research Council, as follows:

1. *Early* (*mild*). Patients with non-specific symptoms, little or no clinical signs of meningitis, no paresis, in good general condition and fully conscious.

2. *Medium* (*or moderate*). All cases between grades 1–3, who were conscious but often irritable, and with meningeal signs present.

3. *Advanced* (*severe*). Patients obviously severely ill, deeply stuporose or comatose or with severe paresis.

Symptoms and Physical Signs

Table 9 shows all the symptoms noted prior to hospital admission. It will be seen that four children were known to have had turberculous lesions previously, a severe blow on the head preceded symptoms in four others by one to four weeks, and erythema nodosum had occurred in two cases within a few weeks of the onset of symptoms.

The first symptom was always a change in the usual behaviour of the child. The school-age child became listless, wanted to sit continually, refused to go to school or out to play. The toddler often became much less active, lay about and fell asleep unexpectedly during the day and was wakeful and fretful at night. During this first stage abdominal symptoms were common. There was vomiting in 82 per cent of cases it was often occasional and not related to food, but tended to occur mostly during the daytime when the child moved about, rather than in bed at night. Sometimes persistent vomiting was the only definite symptom. The appetite was always capricious and the bowels usually constipated. Abdominal pain was the principal symptom in 18 of the 84 cases, so that this diagnosis should always be considered in the differential diagnosis of obscure abdominal pains of sudden onset in childhood. Diarrhoea occasionally accompanied the pain and ketosis was often present.

The temperature had been taken in only 25 per cent of the children and in all these it was found to be raised at some time during the day, usually to 100°–101°. Sixteen children had fits prior to admission, but in most of these the fit heralded a later stage of the disease and was never a very early symptom. Cough was present in a few cases; in seven children there had been a sudden onset of paralysis but, like convulsions, this always occurred in the second or third stage. In taking the history of such cases, a search for possible tuberculous contact, which was often only obtained on direct questioning, was helpful in arousing suspicion of this disease.

Clinical Examination. In the first stage of the disease the child was usually rather listless and quiet, but perhaps slightly miserable and irritable, often with a sallow skin and flushed cheeks. Wasting at this stage was moderate or absent. Frequent sighing was often noted and the pupils tended to remain dilated. Temperature or pulse were always slightly raised. It should be emphasized that there were no other signs of meningeal disease.

When the child reached the second stage, irritability was greater, but consciousness was preserved and typical signs of mild meningismus were present. In contrast to purulent meningitis neck stiffness was slight. The child tended to lie very still and often curled up. Diagnosis was now usually obvious.

The third stage cases were all stuporose or restless and mentally confused. In most cases speech had gone, wasting was severe and the child was febrile, with marked meningismus and incontinent. Often it was unable to swallow and occasionally paralysis of the face, oculomotor muscles or limbs was present.

Results of Treatment

Although it is outside the scope of this paper to discuss treatment, it should be emphasized that the results of treatment in this disease depend so much on the diagnosis in the first or early second stage. In the Ministry of Health Streptomycin Trials, the overall survival rate was 28·6 per cent but in early cases this figure rose to 56·5 per cent. In our much smaller series, only one early case and seven second-stage cases died and survival rates of 86–90 per cent have been reported more recently by Mackay (1954) Manchester and Lober (1954) Sheffield.

SEQUELAE OF MENINGITIS

Unfortunate sequelae were occasionally seen in cases who recover from both purulent and tuberculous meningitis (see Table 10). In the series of cases of purulent meningitis, eleven children show some after-effects, all of whom were infants or children under three years of age. One child died from resulting hydrocephalus six months after

infection was cured. Two children have been left with severe cerebral palsy and mental impairment, and two others have slight residual spastic hemiplegia. Two have developed epilepsy and two are severely deaf but otherwise normal; mild squints are present in two children.

The patients who survived tuberculous meningitis have now been followed up for several years, twelve of these cases started treatment in 1947 and on the whole the long-term results are very gratifying. Only three cases relapsed after apparent cure. An attack of measles precipitated the relapse in two, one of whom unfortunately died quite rapidly, the other two were successfully treated again and have remained well and free from sequelae.

Mild residual spastic hemiplegia is present in one child but is not severe enough to interfere with normal activity, and one child has a unilateral lower facial paralysis. Deafness has been the most serious problem, five children who were treated with dihydrostreptomycin have become severely deaf, one child moderately and another slightly deaf. This drug has since been discontinued for treatment.

As far as has been ascertained up to date the mental capacity of the forty-four recovered children does not seem to have been affected. Several girls are now employed in full-time clerical work, one boy is in the merchant navy, two girls have gained grammar school scholarships and another has won a free place to the Mary Haire Grammar School for the partially deaf.

Since the discovery of antibiotics a revolution has taken place so that these diseases, which only twenty years ago were almost untreatable, now have mortality rates of less than 20 per cent. Even these deaths are a reproach to our profession and a spur to greater effort, since by earlier diagnosis it should be possible to reduce them to negligible figures.

It is popular to believe that for advance in diagnosis an elaborate paraphernalia of apparatus and tests is necessary, but in meningitis, as in many other diseases, the secret of success lies still in the fundamental skill of clinical observation which every doctor has in his power to use, the searching eye to look at the child's face, the listening ear to hear its cry and the mother's story, and the sensitive figure to palpate the fontanelle.

I should like to acknowledge with grateful thanks the help of all those physicians on the staff of United Bristol Hospitals and Southmead Hospital who have allowed me to examine the records of their cases for the purpose of this paper and particularly to Professor Perry, Professor Neale and Dr. John Apley.

REFERENCES

Hutchinson, R. and Moncrieff, A. (1944): *Lecture on Diseases of Children*, ninth edition, p. 3. London.

Henoch, E. (1889): *Lectures on Children's Diseases*, Vol. 1, translated from fourth edition by John Thompson, pp. 336–349. London.

Sheldon, W. (1946): *Diseases of Infancy and Childhood*, p. 502. London.

Alexander, H. E. (1947): *Advances in Paediatrics*, Vol. 11, p. 129. New York.

Ounsted, C. (1951): *Lancet*, Vol. CCLX, p. 1245.

Mackay, R. I. (1954): *Archives of Diseases in Childhood*, Vol. 29, p. 366.

Lorber, J. (1954): *Archives of Diseases in Childhood*, Vol. 29, p. 366.

TABLE 1

Purulent Meningitis

Total cases	105	
First month	14 cases	10 deaths
$\frac{1}{12}$–1 year	46 ,,	8 ,,
1–3 years	24 ,,	3 ,,
3–14 years	21 ,,	1 ,,

TABLE 2

Types of Meningitis (age 1 month to 14 years)

Organism:	Cases	Died
Meningococcus	37	1
H. influenzae	26	2
Pneumococcus	15	6
B. coli	2	2
Streptococci	1	—
Staphylococci	2	1
Unidentified	8	—

TABLE 3

Type of Meningitis—Age Distribution

Age	Meningo-coccus		*H. influenzae*		Pneumo-coccus		*B. coli*		Strepto-cocci		Staphylo-cocci		Un-identified
	Total	Died	Total	Died	Total	Died	Total	Died	Total	Died	Total	Died	Total
First month	—	—	—	—	—	1	7	6	4	2	—	1	1
1 month to 1 year ..	21	1	8	1	8	3	2	2	—	—	2	1	5
1–3 years ..	11	—	10	1	3	2	—	—	—	—	—	—	—
3–14 years ..	5	—	8	—	4	1	—	—	1	—	—	—	3

TABLE 4

Neonatal Meningitis

Case	Age (in days) and type of delivery	First symptoms	Signs	Organism	Result
1	11 Forceps	Fretfulness	Pyrexia	Purulent C.S.F. culture, sterile	Lived: no sequelae
2	6 Normal	Incessant whining, intermittent cyanosis	Grey pallor, bulging fontanelle, pyrexia	Streptococcus	Lived: residual squint, normal otherwise
3	9 Premature	Refusal to feed, pyrexia	Very irritable, rigid	*B. coli*	Lived: no sequelae
4	28	Off food, pyrexia	Convulsions, fontanelle full	*Streptococcus viridans*	Lived: normal
5	9 Breech, premature	Lethargy, pallor, cyanotic attacks	Pyrexia	B. haemolytic streptococcus	Died
6	7 Breech	Lethargy, subnormal temperature	Grunting respirations, bulging fontanelle	*B. coli*	Died
7	5 Early, ruptured membranes	Irritability, subnormal temperature	Rigidity	*B. coli*	Died
8	4 Normal	Cyanotic attacks, pyrexia	Slight head retraction	*B. coli*	Died
9	5 Normal	Cyanotic attacks, pyrexia	Full fontanelle, grunting respirations	*Streptococcus agalactiae*	Died
10	28	Anorexia, coryza	Pyrexia, twitching	Pneumococci	Died
11	21	Screaming, drawing legs up, refusal to feed	Pallor, dehydration	*B. coli*	Died
12	21 Premature	Irritability, rigidity	Pallor, bulging fontanelle	*Staphyloccus aureus*	Died
13	2 Membranes ruptured 1 week	Cyanotic attacks, pyrexia	Grey, limp	*B. coli*	Died
14	2 Premature, forceps	Irritability, sudden collapse	Respiratory irregularity, grey	*B. coli*	Died

Un-identified 1 5 — 3 Total

TABLE 5

Purulent Meningitis—Symptoms before Admission

(Figures refer to number of cases)

	1 month to 1 year	1–3 years	3–14 years
1. Irritability and fretfulness	24	10	1
2. Vomiting	22	14	16
3. Anorexia	24	7	5
4. Lethargy	17	13	10
5. Headache	—	—	13
6. Convulsions	9	8	3
7. Rash	9	4	4
8. Pyrexia	3	6	5
9. Coma	1	2	—
10. Altered respirations	3	1	—
11. Other	2	—	2
Total cases	46	24	21

TABLE 6

Purulent Meningitis—Condition on Admission

Signs	1 month to 1 year	1–3 years	3–14 years
Pallor	33	17	13
Meningismus	30	20	21
Stupor or coma	20	14	13
Convulsions or twitching	12	2	2
Total cases	46	24	21

TABLE 7

Purulent Meningitis—Deaths

No. and age	Organism	History	Admission	Remarks
1. 6 months	Meningo-coccus	20 hours. Vomiting and listless	Coma. Severe shock	Died 7 hours after admission. Supra-renal haemorrhages
2. 6 months	*H. influenzae*	14 days. Fever. Intermittent vomiting. Crying + Coryza	Coma. Convulsions. Neck rigidity	Died 4 days after admission
3. 2 years 8 months	*H. influenzae*	7 days. Coryza. Staring. Refused food. Only vomited once.	Coma. Severe shock and dehydration	Died 6 hours after admission
4. 3 months	Pneumo-coccus	? 7 days. Anorexia. Vomit. Whimper-ing. Coryza	Moribund	Died just after admission
5. 7 weeks	Pneumo-coccus	Vomit. Anorexia. Pallor. 2 days	Convulsing +++ Grey. No neck rigidity. Fontanelle *not* +	Died 6 hours after admission
6. 10 months	Pneumo-coccus	2 days. Anorexia Grunting.	Pallor — Semi-conscious	Died 12 days after admission
7. 14 months	Pneumo-coccus	14 days. Irritable and Fever. Convulsions	Coma. Shock. Neck stiff. Convulsing	Died 6 hours after admission
8. 14 months	Pneumo-coccus	5 days. Irritable 2 days. Anorexia and drowsy	Coma. Twitching. *No* stiff neck. Respirations irregular	Died 12 hours after admission
9. 5½ years	Pneumo-coccus	3 days. Head injury. Headache. Vomiting. Convulsions	Convulsing. Temp. 105°. Stiff neck	Died 18 hours after admission
10. 7 weeks	*B. coli*	3 weeks. Limp and staring. Convulsions and vomiting 3 days	Moribund. Bulging fontanelle	Died few hours after admission
11. 6 weeks	*B. coli*	Rising respiration rate and later convulsions	Fontanelle tense. Neck rigidity. Greyish pallor	Meningitis was a terminal event in a grossly abnormal baby with multiple congenital abnor-malities including imperforate anus
12. 11 months	*Staphylo-coccus aureus*	Refusal of food. Vomiting. Lethargy	Moribund	Subdural haema-toma since early infancy. Grossly re-tarded child men-tally. Operation was performed 4 weeks previously

TABLE 8

Tuberculous Meningitis

Total Cases	84
Recovered	44
Died (Treated)	24
Died (Untreated)	16

TYPE OF CASE (TREATED)

	Early		Medium		Advanced	
	Lived	Died	Lived	Died	Lived	Died
Under 1 year ..	2	—	—	2	3	1
1–3 years	2	—	6	4	3	5
Over 3 years ..	7	1	18	1	3	10
Total cases ..	11	1	24	7	9	16

TABLE 9

Tuberculous Meningitis

Total Cases: 84

Symptoms before Admission:

Vomiting	69	
Lethargy	58	
Anorexia	33	
Headache	32	
Abdominal pain	18	
Pyrexia	21	
Fits	16	
Cough	17	
Paralysis	7	(4 eyes, 2 arms, 1 face)
Other T.B. lesions	4	
Head-injury history	4	
Erythema nodosum	2	

TABLE 10

Sequelae

PURULENT MENINGITIS (11 PATIENTS)

	Age of onset:	Type of meningitis:	Residual signs:
1.	5 days	Streptococcal	Mild strabismus
2.	6 weeks	Meningococcal	Severe hydrocephalus; died later
3.	11 months	Meningococcal	Occasional major fits
4.	11 months	Meningococcal	Mild spastic hemiplegia
5.	15 months	Meningococcal	Mild strabismus
6.	7 months	*H. influenzae*	Major fits
7.	9 months	*H. influenzae*	Severe spasticity, blind Mental defective
8.	18 months	*H. influenzae*	Deafness
9.	2 years	*H. influenzae*	Deafness
10.	10 months	Pneumococcal	Spastic hemiplegia
11.	2 months	Unidentified	Spastic diplegia—mental defective

TUBERCULOUS MENINGITIS (9 PATIENTS)

1.	7 years	Spastic hemiplegia
2.	4 years	Left facial paralysis
3.	6 months	Severe deafness
4.	4 years	Severe deafness
5.	7 years	Severe deafness
6.	9 years	Severe deafness
7.	12 years	Severe deafness
8.	5 years	Moderate deafness
9.	5 years	Slight deafness

MENINGITIS

BY

A. M. G. CAMPBELL, M.D., F.R.C.P.
Consultant Physician, United Bristol Hospitals

INTRODUCTION

Inflammation of the meninges and brain in the past was always considered such a serious and fatal disease that its early diagnosis was not stressed sufficiently. Since the introduction of antibiotics it has been shown that mortality figures are in direct proportion to the delay in diagnosis and treatment. Therefore, the early diagnosis of meningitis and establishment of the organism, which has infected the brain, are vital.

Infection of the meninges may be *via* the blood stream, as is predominantly the case in meningococcal meningitis, or by a direct spread from neighbouring structures, such as infected bone or sinuses. Meningitis may also be due to chemical irritation from spinal anaesthetics or from virus and spirochaetal infection.

The onset of a meningitis can be extremely acute and is accompanied by generalized severe headache but, in particular, the headache is very often localized to the back of the neck. All the signs of a meningeal irritation depend on a reflex muscle spasm arising from stretching inflamed spinal roots, either in the cervical region accounting for neck rigidity or in the lumbar region accounting for Kernig's sign, a sign which does not allow of extending the flexed knee with hip flexion. It must be emphasized that any irritation of the meninges such as that caused by blood in a sub-arachnoid haemorrhage or by increased intracranial pressure associated with a cerebral tumour, may produce these signs. Again in certain febrile illnesses, particularly pneumonia and typhoid, and occasionally tonsillitis, these signs may be positive and account for the so-called case of meningism, which is thought to be due to a fall in the osmotic pressure of the serum and increased production of cerebrospinal fluid, but is not accompanied by cellular increase in the fluid.

The diagnosis of meningitis from other irritative lesions of the meninges can only be made by lumbar puncture, which is absolutely essential in any doubtful case of this kind, not only for diagnosis but to establish the organism and its sensitivity.

The early symptoms of meningitis differ slightly in children and adults. Dr. Corner has enlarged very fully on the difficulty of diagnosis in children and emphasized that headache is not such a predominant symptom in childhood as it is in adults. A certain number of adult cases will arrive in coma so that other causes of coma are a differential diagnosis in this disease. Other cases present with epilepsy or with some other odd symptoms which I will mention later in connection with tuberculous meningitis; one pneumococcal meningitis presented with sciatic pain.

All damage to the brain whether it is infective or traumatic leads to intellectual impairment during the course of the illness. In many people this intellectual impairment strikes at the very base of their personality and, even after recovery from the primary condition, they may become unduly worried about the question of brain damage and often a secondary anxiety state is grafted on to any organic disease which remains. If the individual previously had a somewhat neurotic personality, he is best treated by careful reassurance and graduated convalescence. It is particularly important to reassure him about his future attainments. These symptoms differ in no way from those shown by anxious people after a head injury.

Other possible complications of any meningitis are adhesions in the arachnoid space, which may produce constriction of the spinal cord, paralysis, and hydrocephalus.

Lastly, epilepsy can follow meningitis just as it can follow a head injury and it can

come on several years after the acute attack; it is usually related to the severity of the initial illness and whether treatment has been instituted early.

MENINGOCOCCAL MENINGITIS

Meningococcal meningitis is largely a disease of children, but it does attack young adults and in particular those living in the crowded conditions which occurs in wars. In the spring months, it can take an epidemic form. This was seen in 1915 and again in 1940. In the R.A.F. in 1940 I was in the position of looking after two wards of meningococcal meningitis. In this series in all we had 54 cases and 2 deaths. One patient died as the result of a fulminating attack accompanied by septicaemia, purpura and haemorrhage into the suprarenals within twenty-four hours of the symptoms developing. In this R.A.F. outbreak, we had two patients with deafness, which is due to the disease infecting the labyrinth. We had one case of herpes zoster supervening on the disease and two cases of meningococcal septicaemia associated with meningitis and producing arthritis.

PNEUMOCOCCAL MENINGITIS

Pneumococcal meningitis is a disease of all ages, but occurs very frequently in adults. Its cause is usually an infection from the mastoid air cells, sinuses or middle ear, but quite a few cases are the result of trauma and a fracture of the inner wall of a sinus allowing infection to spread to the meninges. Another important cause is pneumococcal infection of the chest or the spread of infection from bronchiectasis or lung abscess. In the last group the occurrence of serious disease both in the chest and in the head undoubtedly account for many difficulties in treatment for many of the cases die as a result of their chest condition rather than from their meningitis, or a combination of both conditions. Sometimes the disease follows operation and two cases recently have followed operation for polypi in the antrum.

Before the days ot antibiotics, pneumococcal meningitis was almost always fatal, but, with the introduction of sulphonamides, the mortality was reduced to approximately 70 per cent, and with the addition of penicillin to the treatment, the outlook has been greatly improved and the mortality has been reduced to between 30 and 40 per cent. Results in certain Units, such as Oxford, have been strikingly good and the mortality has been as low as 24 per cent. The difficulty in comparing mortality figures in this disease is the association of other diseases, which is quite common with pneumococcal meningitis in elderly individuals.

The occurrence of this type of meningitis in the old-age group is quite striking and within the last year, I have treated two cases of this disease in patients over sixty-five years of age. The first recovered and the second died. The second case was sent to hospital late because the doctor imagined that the epileptic fit which the patient presented with, was due to cerebral vascular disease and not due to meningitis.

The rapidity of the onset of this type of meningitis is quite remarkable. For example, the wife of a doctor went to bed with ear-ache one night. She complained of ear-ache and headache the following morning and by 1.0 p.m. was found unconscious, with severe meningeal symptoms. Treatment with intrathecal penicillin was instituted at 4.0 p.m. and she made an uneventful recovery.

The second personal case was that of a boy of sixteen years of age, who was knocked on the head in the frontal region whilst playing football at 3.0 p.m. At 6.0 p.m. he complained of headache and was admitted to the Royal Infirmary, where a lumbar puncture was performed at 8.30 p.m. He was already semi-comatose and was found to have a purulent pneumococcal meningitis six hours after the blow. He also made an uneventful recovery. Unless treated promptly these fulminating cases will succumb very rapidly, so that early diagnosis is absolutely essential.

The question of therapy for pneumococcal meningitis does not intimately concern general practitioners. It suffices to say that sulphonamides and penicillin are both

necessary; and penicillin should be given systemically and intra-thecally. Whilst meningococcal meningitis does extremely well on sulphonamides alone, penicillin is the sheet-anchor of the treatment of pneumococcal infections. The newer antibiotics are quite unnecessary unless the organism is resistant to penicillin, and it has actually been shown the aureomycin given with penicillin produces a worse recovery rate than penicillin alone.

Where the meningitis results from trauma due to infection entering through a dural tear, a fascial repair by a neurosurgeon is necessary to prevent relapses.

Sometimes the cause of infection remains unknown even after extensive investigations, and one such case in Bristol had six relapses in seven years, all responding to penicillin. This patient can now diagnose his meningitis within an hour of its onset. Another patient had a dural repair on one side and then relapsed and was found to have a tear on the other side.

Any therapy must be maintained until the cerebrospinal fluid regains near normality, and a lumbar puncture must be done after therapy has ceased to see that there is no recurrence. Sometimes loculi of infection occur in the ventricles and account for persisting unresponsive infections.

Rarely an abscess may develop in the brain and I would stress the fact that meningitis can accompany a localized abscess. If a meningitis fails to respond to adequate therapy or localizing signs of brain damage develop, particularly with a raised protein, an abscess should always be suspected. Air pictures, arteriography and electro-encephalograms should then be done.

It will be seen that any case of meningitis needs the most careful observation and control. The position in these cases is always changing from day to day and any development of new symptoms will have to be met by new measures to avoid complications or to deal with these complications. Treatment, therefore, has become a highly specialized technical procedure demanding the co-operation of the bacteriological laboratory and the clinician. It cannot, and should not, be carried out at home. In any Unit treating meningitis, the results of treatment of any form are very much rosier than they were ten years ago.

It remains to say one word about pyogenic meningitis arising from lumbar puncture. It is unfortunately true that pyogenic meningitis sometimes arises as a result of lumbar puncture or the introduction of a spinal anaesthetic. The sterility of lumbar puncture needles and lumbar puncture set is absolutely essential to avoid these serious accidents. If a patient persistently runs a temperature after a lumbar puncture or the neck is unduly stiff, the possibility of the development of a meningitis must always be considered.

PREDOMINANTLY LYMPHOCYTIC MENINGITIS

Meningitis in which the cells in the cerebro-spinal fluid are predominantly lymphocytes can be classified as follows:

Due to organism:
1. Tuberculous meningitis
2. Syphilis
3. Leptospirosis

Due to virus:
1. Poliomyelitis
2. Mumps
3. Choriomeningitis
4. Glandular fever
5. Herpes simplex and zoster
6. Unknown cause

TUBERCULOUS MENINGITIS

Dr. Beryl Corner has dealt with the disease in children, and it only remains to say that in adults the basic symptomatology and treatment are the same. The diagnosis

can be difficult for the onset is very often insidious and it is not always realized that the disease can occur at any age. Examination of the fundi for choroidal tubercles is more important and easier in an adult than a child. Chest X-rays in adults often show that the disease is part of a miliary process.

One patient presented at the Bristol Eye Hospital with an ophthalmic herpes, which was a symptomatic manifestation of a severe tuberculous meningitis at the age of sixty-three.

On examination of the cerebrospinal fluid, the finding of the organism, and persistently low sugar rates are characteristic of the disease; but a low sugar in the cerebrospinal fluid is highly suggestive and treatment should be instituted with streptomycin, isoniazid and, if necessary, P.A.S. At this point, I would like to add that I am quite certain that intrathecal treatment should be persisted with unless there are very good contra-indications. The cases that have not been treated with intrathecal streptomycin on the assumption that the introduction of isoniazid has made this unnecessary have not been followed up sufficiently long to assess whether the relapse rate is as satisfactory as it is in those cases treated intrathecally.

We have had two interesting cases of pregnant women with tuberculous meningitis in the Bristol series. The first was treated in 1948–49; unfortunately she relapsed after delivery with acute pulmonary spread of infection and died. Her child lived and is perfectly fit and well at the moment, and I might quote a letter from Dr. Hartley received recently.

"The baby was transferred from Southmead Hospital to the Babies Home and was under my care during the first two years. She made uninterrupted progress being physically of very good standard and hearing and mentality in every way normal. Skin tests for T.B. during this period also remained negative.

"After two years she left the home, but I have kept in touch with her and have seen her on many occasions, and she is still very well and now at school."

This case is important as shown by the fact that the child did not suffer as a result of her mother's disease or from the treatment given during pregnancy, and the labyrinth of this child is quite normal.

We have recently treated a patient in the Bristol Royal Infirmary who was satisfactorily delivered by Professor Lennon (by a Caesarean section) of a baby weighing 8 lb. Both mother and child are now perfectly well.

Occasionally tuberculous meningitis is accompanied by endarteritis of the brain and cerebral thrombosis is apt to occur with hemiplegia, which may mask the tuberculous process.

You may ask if treatment entirely eradicates this disease. In the best hands there is recovery of 50 per cent of cases of tuberculous meningitis, but a certain number of these may relapse and unfortunately there is always a slight risk of a relapse in a case of tuberculous meningitis. On the other hand, Dr. Macrae at Ham Green Hospital treated a man in the fifties, who died eighteen months later of heart disease and a post mortem carried out at Exeter showed no evidence of tuberculosis of the brain.

We can now offer these sufferers a very reasonable hope of a satisfactory life and if care is used there is certainly no danger from the treatment itself. I have included tuberculous meningitis in the lymphocytic group although many cases show a polymorph response in the fluid, particularly in the early stages.

SYPHILIS

This disease presenting as an acute meningitis and as part of a meningo-vascular syphilis is rare. The onset is usually more chronic and does not cause confusion with an acute meningitis, but occasionally syphilis can present in this way, resembling any other acute meningitic process.

In the last seven years, I have seen one such case. The disease was suggested by focal signs, ocular motor palsy and Argyll Robertson pupils, and confirmed by the Wassermann reaction, which was positive in blood and fluid.

LEPTOSPIROSIS

Leptospirosis occurs round Bristol in the form of Weil's disease and Canicola fever. Weil's disease is usually contracted as a result of bathing in stagnant pools, which are frequented by rats, and in any meningitis in which there is a history of recent bathing, this infection should be suspected. The diagnosis is also suspected by suffusion of the conjunctivae and the development of jaundice. Canicola fever, which is contracted from dogs, is not such a severe disease and predominantly shows a lymphocytic meningitis. Contact with a sick dog, or for example, in one case, bathing in a pool in which there was a dead dog, may be responsible for infection. About fifteen cases occur a year in Bristol and both types can be diagnosed by agglutination reactions.

ASEPTIC MENINGITIS

Meningitis in which no bacterium is to blame may be called "Aseptic". Wallgren has classified the criteria for aseptic meningitis as follows:

1. Acute meningeal onset. 2. Predominantly lymphocytic meningitis. 3. Absence of bacteria in the cerebrospinal fluid. 4. The usually short, more benign type of illness. 5. The absence of evidence of sinus, aural, septicaemic or traumatic origin.

POLIOMYELITIS

By far the most important meningitis in this group is, of course, poliomyelitis and where paralysis does not occur the diagnosis is not always easy. One should always suspect the possibility of poliomyelitis in every case of lymphocytic meningitis, particularly when there is a history of an alimentary disturbance or a sore throat with the typical root pains. Lumbar puncture will not always help in the diagnosis. In a proven case of paralytic poliomyelitis it is unnecessary and possibly harmful; but in a doubtful case it is justifiable to exclude the possibility of bacterial meningitis.

The diagnosis of poliomyelitis will, of course, be thought about in a summer epidemic period and in particular in the months of July to October. But a thorough examination of the patient and a careful history is always necessary; in three cases that have previously been diagnosed as poliomyelitis, I have been able to reassure the relatives that the disease was merely mumps. In the first case, a recent contact with mumps proved helpful and in the other cases, slight enlargement of the parotid glands and testicular pain showed that the correct diagnosis was mumps and not poliomyelitis.

HERPES SIMPLEX AND GLANDULAR FEVER

Both herpes simplex and glandular fever may be responsible for lymphocytic meningitis. In glandular fever, the Paul Bunnell test and blood count may be of help. Herpes can only be diagnosed as a result of agglutination reactions.

In a survey of lymphocytic meningitis occurring in the American Forces over a period of ten years, it was shown that 10 per cent of these cases could be accounted for by mumps; 10 per cent by leptospirosis; 5 per cent by herpes; 10 per cent were probably lymphocytic choriomeningitis, (a virus disease which many think is contracted from mice); the remaining 65 per cent could not be diagnosed on pathological grounds and were probably non-paralytic poliomyelitis. All that can be said is that if a lymphocytic meningitis occurs in the winter, it is less likely to be due to poliomyelitis than if it occurs in the summer. A careful history and a careful clinical examination will help in the diagnosis of most cases, while a further doubtful case may be diagnosed by referring blood and cerebrospinal fluid to the Central Virus Research Laboratories under the direction of Dr. McCallum of Colindale. Unless the diagnosis of poliomyelitis is established, every effort should be made to find the cause of the meningitis by pathological tests, as I have indicated, and by examination of the cerebrospinal fluid.

In conclusion, let me stress again that the most important point in meningitis is the need for early diagnosis and early treatment. The antibiotics, which we have at our disposal, will be wasted unless the diagnosis is made early and the treatment is instituted early and for sufficiently long in each case to control the disease. Every practitioner should be on the lookout for the very suggestive signs of neck rigidity or root irritation, and when in doubt the case should be referred to a hospital for specialized opinion. The recovery rate in most cases of meningitis, whatever their cause, is so good that the whole outlook of this once very serious condition has been changed in a decade.

WHAT THE G.P. WANTS FROM A CASUALTY DEPARTMENT

BY

A GENERAL PRACTITIONER

Casualties coming in direct. The general practitioner will expect that cases will be admitted when necessary. When the injury is such that it can be dealt with and the patient discharged, the doctor expects to have a note sent with the patient from the casualty officer briefly stating the injury, X-ray findings, what has been done, and the line of treatment recommended. Where the seriousness of the case demands it, the doctor should be informed about it by 'phone.

It should be unnecessary for the doctor to have to write a letter referring his patient to Out-Patients, if that patient has sought advice himself at the Casualty department.

Casualties referred by the family doctor. If the injury can be dealt with at once, the G.P. expects a report on lines similar to that given for direct casualties. If he has expressed a wish for the case to be detained or admitted the G.P. expects that this will be done. No-one below the status of a registrar should take the decision to refuse admission in such circumstances. On the other hand often the single visit to Casualty would suffice, and chemotherapy, dressings and removal of stitches could be left to the family doctor, who has the district nurse to call on. This would ease things at the department, without unduly loading the doctors individually. In these days of litigation, many cases are referred by practitioners for X-Ray examination to protect himself against legal action, and a great deal of unnecessary radiography has to be accepted. Prompt reports on the wet-films are therefore much appreciated.

Septic fingers and hands. Finger sepsis can lead to serious and prolonged disability, and the family doctor is glad to have available such a service with adequate anaesthesia and skilled surgery.

Cold minor surgery, and so forth. Individual doctors vary in their desire to engage in minor surgery such as wens, and lipomata, and since the financial barrier to the hypochrondriac's access to the doctor has been removed, time for minor surgery is hard to find, and some doctors and their patients are grateful to have easy access to Casualty for this sort of treatment.

The time spent in the casualty department should be reasonable. Patients tend to complain when they have no ground to do so and here the G.P. can help by explaining to patients why X-Rays and treatment take time.

Accommodation. Patients should have proper privacy for undressing and should be spared any unpleasant experiences such as seeing other people's wounds being dressed.

The family doctor has at his disposal in Bristol a casualty department at the Royal Infirmary which meets his requirements very well. This seems to be attributable to the appointment by the Board of Governors of a casualty surgeon of consultant status. The lack of a casualty department in the General Hospital is still felt; an eight- or twelve-hour service at the B.G.H. would be a help to south Bristol and would relieve pressure at the B.R.I. For an industrial city of over half a million, the present casualty departments at B.R.I., Southmead and Cossham-Frenchay hardly meet the need; and the Regional Board should not continue to leave unused the commodious department at the B.G.H.

CASUALTY DEPARTMENTS

BY

HERBERT K. BOURNS, M.B., F.R.C.S.

Consultant Surgeon in Charge of Casualty Department, United Bristol Hospitals

There have been considerable changes in every branch of medicine since the inception of the Health Act, and in no branch is this more obvious than in the casualty departments of our hospitals throughout the country. There are many reasons for changes and some of the main ones are discussed under three main headings.

Firstly the public have changed in their attitude to the hospitals. The nearby institution is no longer their hospital in which they take a pride and for which they organize collections and fairs. It is now a part of the National Health Service and the doctors and nurses who work there are State employees. Since it is no longer their hospital but a government responsibility they have little respect for it. Since they now have no direct financial responsibility or interest, it is a concern in which the best should always be available, no matter what it costs. Many, of course, believe that the major portion of their weekly contributions under the National Health scheme is directed to the hospitals and doctors. For this reason they do not see why there should be any need for economy in either equipment or personnel. The general public now regard the casualty department as a convenience to be employed at will, and to be criticized freely. Any shortcomings which in their opinion exist, must be corrected regardless of what this may involve in extra staff or money. Judgement is passed hastily and without any consideration of any difficulties with regard to either staff or accommodation.

Secondly the doctors view the casualty department in a different light. There is no point in a doctor under present-day conditions of general practice, carrying out minor surgery or treating extensive septic conditions which require incisions and dressings. He is not repaid either by the State or the patient for his trouble, and he only adds further anxiety and burdens to his already over-full day. The doctor too has to contend with long waiting lists for out-patient appointments. Every patient wants to see a specialist. A case may not be acute but a second opinion may be of little value if it can only be obtained in two months' time. The G.P. adopts the simple expedient of sending such a patient to the casualty department. A second examination is made by a doctor and this is reassuring both to the doctor and the patient. The idea in the mind of the public that a hospital doctor is always better than their own general practitioner still exists and the youthful face of the junior hospital officer does not seem to disturb this faith.

The third factor which is bringing about a great change in casualty services is the large number of industrial accidents which require immediate treatment of a high standard. Minor injuries require expert attention if they are not to have major consequences. The treatment accorded to a lacerated finger in the first twenty-four hours may make or mar the final result. Full function may not be achieved because the primary treatment was inadequate and ill-conceived. With the increase in mechanization and the shortage of skilled workers, the numbers of accidents are greater and the results of inexperienced treatment more disasterous than ever before.

SERVICE MUST BE ADEQUATE

It is easily discerned, then, that a casualty department is nowadays a place where expert opinion and first-class technique should always be available. Anything less than this is no longer justifiable. To turn to each class of person which utilizes the casualty service of the local hospital, and to lay down strict rules of procedure which must not

be transgressed is not to recognize their difficulties and to alienate good will and co-operation. To turn away everyone who cannot justify his presence on all counts is to try to put the clock back, and does not relieve us from the responsibility of producing a much improved service.

Reorganization of casualty departments must be carried out everywhere. Consultants in hospital and doctors in practice must recognize this, but they must also both acknowledge the difficulties during the present period of change, the rate of which is governed by many factors outside the control of medical administrators.

A casualty department must be organized to cope with patients when neither the numbers nor the diseases are known in advance. The notice " House Full" can never be placed over a closed door, but neither should the notice "Welcome" be placed upon the threshold. To cover a busy department so that immediate attention can be accorded to all, means over-staffing unless the number of attendances per hour can be found to follow a rough pattern. In a recent survey at the Bristol Royal Infirmary this was done, and the hourly attendances were noted for a week. It was apparent that attendances did follow a rough pattern with the periods 9.0–10.0 a.m., 10.0–11.0 a.m. and 2.0–3.0 p.m. showing about equal and maximum attendances over all other periods. The introduction of "block" appointment systems for all re-attendances is essential in the future but requires a considerable clerical staff, and accommodation large enough to allow for separation of both new and old cases. Planning will help, but even when numbers are accurately forecast, the type of case which will attend at any time cannot be predicted. One morning may see three serious accidents with the resultant severely injured to be treated. One casualty officer is thus immobilized for a long time and the cases to which he normally attends will inevitably have to wait and so accumulate because they cannot be turned away.

It will be appreciated then that staffing presents a difficult problem, but in another respect the variation in the type of cases may alter the whole demand for medical attention and time on any particular day. Many cases require operative treatment beyond the skill of either nurse or student. To undertake a skin graft, remodel a partially amputated finger or remove a small skin lesion from an important region is a short procedure easily performed under local anaesthetic, when the equipment and the medical staff are available. But a Casualty Officer will put a strain on his own skill and the patient's understanding if he tries to fit these cases into his daily routine. To pass such cases on to the staff of the hospital is uneconomical in both time and beds. The surgical team on duty may be in the middle of a busy out-patient session or a long operating list. The patient cannot be attended to until a convenient moment arrives and this may not occur until many hours have passed. He may even have to be admitted because the operation is not carried out until a late hour. Delays such as these are bad for the patient and his lesion and for the hospital. The casualty services must be equipped for such cases within its own resources.

JUDGEMENT NEEDED BY CASUALTY STAFF

We have already stated that both patients and, to a lesser degree, doctors use the casualty department because its doors are always open. Many patients plead ignorance of their own doctors' surgery hours. Many state that they cannot wait until the next surgery and again many reply, when questioned about attending the hospital, that they do not wish to bother the doctor with such a trivial matter! All patients have to be examined and assessed before they can be safely sorted out. Many non-urgent cases not referred by doctors occupy valuable time which should be devoted to the treatment of emergencies. Medical officers must be available to deal with these cases because a hospital cannot refuse treatment to any person until his category has been established on competent medical advice. For the general practitioner the despatch of a patient to the casualty department may be the only method by which the patient can be presented quickly to the hospital.

Again, an unknown person collapses in the street or at work. He is taken to the

hospital at once. He is directed to the casualty department. He may present a most difficult problem which may be quite outside either the experience or knowledge of a junior medical officer.

A case of multiple injuries is brought in. There may be indefinite signs of both head and abdominal injuries. It requires a doctor of great experience to sort out such a case and to try to balance the contesting demands in diagnosis and treatment.

Who is to deal with all these cases in the hospital?

Will the general practitioner of many years' standing who is worried about a patient be satisfied to have a junior medical officer as a second opinion? Will the hospital authorities and the Minister of Health be content with an organization in which the most junior medical man is faced with the most difficult and the most urgent problems? There are other channels of disposal open to the doctor, such as domiciliary consultation, but these are not always easily available. There are no other methods available to the hospital authorities other than a great increase in the number of consultants and registrars, if the urgent cases are not to be directed to the casualty department.

In staffing a department, therefore, there must be medical staff sufficient not only to deal with the daily routine, but adequate to cope with peak periods and times of unexpected emergencies. This will mean that routine work can carry on quietly and without serious dislocation at times of stress. In addition there must be a medical officer available to carry out the surgical procedures for which all casualty departments must be equipped. Otherwise the junior medical staff will be so pressed for time that both they and their work will suffer, and very soon reach a dangerously low standard. Fatigue will come on earlier and with more serious results. Mental exhaustion is a most important factor. In any business, experts are continually watching the personnel so that their work is balanced and in many cases frequent changes and liberal breaks are given in order to promote efficiency, production and accident-free work. In a Casualty Department, house officers must not be driven so that they become fatigued. There is a continuous demand for acute and keen mental discernment and, in order to maintain a high level of efficiency, liberal time for leisure must be afforded to all the staff.

NEED FOR SENIOR MEDICAL OFFICER ON DUTY

With regard to the senior medical staff required, it is quite impossible for the hospital to provide cover for serious cases from the resources of visiting specialists and registrars. They have their daily routine which fully occupies their time, and they cannot be expected to run the casualty department at irregular intervals during the day. The examinations of a case of either collapse or serious injury takes at least half an hour, and investigations such as an X-ray examination considerably prolong this period. It is obvious, therefore, that a consultant or registrar or both, must be on the casualty staff. His experience will be invaluable in teaching and supporting the junior house officers. His opinion on subacute cases about which a medical colleague in general practice is worried, will be available and useful. His skill will be present to perform all manner of surgical procedures which many may conceive as minor, but which are now generally recognized as being as worthy of medical skill as an inoperable cancer or an incurable nervous disease. His confidence will be equal to the most difficult and truculent patient who will have to be firmly and correctly treated without medico-legal repercussions of a serious nature. Lastly the presence of a consultant will make for a restoration of confidence in the casualty departments of our hospitals.

The senior man must not only be easily available, but must be present in the department, and for two main reasons. Firstly, when a junior medical officer makes a mistake it is rarely because he is slack but nearly always because, in his inexperience, he does not recognize the seriousness of a case. Secondly, if a second opinion is not readily available to him, he will not seek it as frequently as he should. In large hospitals this consultant cover can and must be provided. In smaller hospitals the consultants or their deputies must be involved, and a return to the rank of Resident Surgical Officer would be a great help. This man can be responsible for and help the Casualty Officer.

NEED FOR BEDS IN CASUALTY DEPARTMENT

Adequate in-patient accommodation is of great and vital importance. Without beds the casualty department will be severely handicapped. Firstly the beds must be available to and under the direct control of the staff of the department. The rate of admission and discharge is much higher than in any other department of the hospital. There will always be cases of head, abdominal and other injuries which, although not serious, cannot be sent home safely. Observation in hospital will be necessary for at least twenty-four hours. Secondly there are many inflammatory lesions in which a short course of intensive treatment at once will be the most effective therapy if time is not to be wasted in stemming the disease and in repairing its effects. Thirdly patients on whom minor surgical procedures are carried out, may require a bed for recovery, and unless there are beds in the department, it may be impossible to find beds for such cases without a great deal of planning. There are many other reasons why beds are an absolute essential and their provision brings relief not only to the casualty department, but to the whole hospital.

SEPARATE CASUALTY OPERATING THEATRE

An operating unit is as necessary as in-patient accommodation in any modern casualty department. Small ill-equipped operating theatres have no longer any place in a hospital where casualty and accident cases are received in any numbers. No operation should be undertaken unless full facilities for all that is found to be required in both time and equipment is available Under present conditions many cases have to wait until an operating theatre is empty before essential and urgent surgery can be carried out. This wait is most undesirable. Open wounds, severed arteries and nerves require immediate treatment. The degree of urgency of all the work carried out in a casualty and accident department is much higher than that affecting the rest of the hospital. It is most unsatisfactory that a young fit person with a serious injury should have to compete for theatre time with an old person with a rupture or some other non-urgent surgical problem. Both require treatment but their demands in time are not comparable. An operating theatre in the casualty department adjacent to the wards will get over this difficulty. In it traumatic cases would have priority, and the clinical state of the patient rather than other non-clinical considerations would decide the time of operation. Only then will the advances made by experimental surgeons be brought to bear in the proper manner on cases of trauma. Only then will the advantages of rapid resuscitation be utilized in the most economical and skilful manner. Time must be made to serve the case and not the other way round.

PROBLEM OF FRACTURES

Cases in which bones are injured attend all casualty departments in large numbers. They are a special problem now that fractures are treated in special units by orthopaedic surgeons. In some hospitals these cases are referred to the fracture department as soon as a lesion is noted and they are taken over by the specialist staff. This at first sight would seem to be satisfactory, but there are two serious objections. In the first place the lesion has to be diagnosed by a casualty officer who has little contact with fractures and no opportunity of increasing his knowledge of the broken parts. He cannot be competent to assess each case as to its seriousness or its urgency; since he does not treat fractures, he does not become experienced in the interpretation of X-rays. A medical man who treats bony injuries obviously gains confidence and keener perception in both minor and major problems. Secondly in many cases of fracture, the broken bone may not be the major issue with regard to management. A department where skin loss, soft-tissue injury and other lesions can all receive equal attention is most desirable. Orthopaedic surgeons engaged in a busy practice cannot easily give equal consideration to all sides of a complicated case. This is, of course, not due to any lack of ability, but to a lack of time and of recent experience. An accident service with

accommodation for cases in which bones are broken and soft tissues are disrupted is the ideal plan. In it there should be facilities for a surgical team which is interested in all aspects of trauma to work. Other authorities may be consulted, but there will be no division of responsibility, and the staff will be familiar with all types of treatment. In such a department there would be ample opportunity for a surgeon to carry out satisfying work, and to advance both his own and general experience.

CASUALTY CONSULTANTS REQUIRE BALANCED WORK: ACUTE AND NON-ACUTE

The immediate treatment of any condition is closely bound up with and cannot be separated from the whole course of therapy. The medical man who examines the case in the first instance should be fully conversant with the plan for that particular lesion. It must be recognized that the senior staff of a casualty department must be able to carry out both definitive treatment and non-acute surgery. The surgeon in charge of casualty must have facilities in the department or elsewhere for non-urgent surgery. Apart from this, no-one can spend a lifetime giving opinions on acute abdominal emergencies if he can never follow up his clinical judgement by operation, nor can he be expected to remain interested in the treatment of burns in the acute phases if they always pass from his care in the very early stages. It is obvious that the casualty consultant's opinion will cease to be of much value if he ceases to be an active member of the profession. To keep his balance he must have an outlet for his training and ability and a means of gaining further experience. Therefore, it is obvious that there must either be two consultants or one consultant with a deputy of considerable experience in the casualty department in order that there should be adequate service throughout the twenty-four hours.

THE FUTURE

Looking ahead, it would seem that our casualty departments must develop into accident units or hospitals. Then all accidents, both great and small, can be directed to the hospital where all will know that there are full and adequate facilities available. Both doctors and patients will regain lost faith and confidence and closer co-operation between the general practitioner, factory doctor, and hospital officer will lead to better treatment and less misunderstanding. Then a case can be treated by all together or each in turn, and each one will know the plan of treatment for the case and will know the aims of his colleagues although all will be working from a different angle. If Accident Centres are evolved, the hospitals will have senior staff who have an interest and work for a lifetime of service, and the doctors will have colleagues in the hospital who will share their difficulties in the accident cases, and in those cases requiring urgent opinion, not falling into the narrow channels which are available at present.

No matter what changes take place, we must all try to see that our casualty departments are kept free from unnecessary and unsuitable cases. The doctor looks to the hospitals for a better casualty service and he must get it, but the hospital looks to him to sort out the patients so that only those who will benefit from immediate treatment are sent up. The public finds the open doorway a great advantage and discouragement alone will not make it turn away. The restoration of faith in the family doctor which appears to be evident, and on the increase, will do more than an overpowering sister or a forbidding doctor. Again closer co-operation between hospitals and doctors will improve matters. The good patients suffer and the bad thrive on a system of medical planning in which one doctor does not know the other. The opportunities for the exploitation of such a system are many when communication between colleagues in different spheres is negligible or absent.

An efficient casualty department is essential, but equally important are the health centres and the surgeries. The central department and the peripheral surgery are complementary, and their closer integration must be the aim of all connected with medical administration of both hospital and general-practice services.

JAUNDICE WITH HEPATIC FAILURE—A CASE ILLUSTRATING DIAGNOSTIC PROBLEMS

A Clinico-Pathological Conference of the University of Bristol Medical School

CHAIRMAN: PROFESSOR T. F. HEWER

Professor Hewer: The woman we are discussing this evening was first attended by Dr. Eastman, her general practitioner.

Dr. F. V. Eastman: This lady was one of the well-known patients—always at the surgery! Until this illness started it had always been a question of " nerves ". It was not until September 1953 that she began to have symptoms of organic disease. She complained her heart was beating fast and of some upper abdominal discomfort. She managed to get over this and I did not see her again until the end of the month, when she was jaundiced. There was no story of running a high temperature. She was in the Bristol Royal Infirmary within a fortnight and four weeks later when she left hospital she was happy and bright. Her jaundice was less but was still present. I was called to see her again early in the New Year and then she was not so well. The previous afternoon she had vomited, she had abdominal distension and I thought she had an intestinal obstruction. I sent her to hospital straight away and was sorry to learn that she died three or four days later, on January 7th, 1954.

Mr. R. V. Cooke: This is a very instructive case and I am sure my medical colleagues will support me when I say that these cases are difficult to diagnose. I remember this problem very well because we spent a long time discussing it on one of the ward rounds. She had been jaundiced for five weeks at least when I saw her first. That is not a very long time as I have known a case go on for three months, even longer, before finally clearing up—without surgery! However, we went into the story very carefully as one can learn much from the history. There was in the history a definite suggestion of the onset of hepatitis. She had not been well before the jaundice became evident and, looking at the chart, there was still a little fever—enough to suggest an infective process. The absence of pain did not mean anything conclusive. When she was in hospital in October she was very jaundiced: it would need to be a severe infective hepatitis to bring about so much jaundice. Surgical jaundice can occasionally be painless even if it it is due to a stone. The liver was palpable two-fingers breadth below the costal margin. Should we proceed to laparotomy or should we resort to various liver function tests? Rightly or wrongly I felt there was not time to wait for a result of such tests; and they might well have been unhelpful because the pathologist should be given a case early if a definite answer is to be given. I should like to hear which of the tests in these circumstances are regarded as valuable and how much value is placed on them. I think they are of very little value unless done early. The surgeons are equally confident and they are often wrong! A cholecystogram was done but even that was not a very great help. The opaque material failed to show up the gall-bladder: that perhaps rather tended to suggest a surgical gall-bladder and I decided we would explore her (on October 28th, 1953). We found the gall-bladder was not enlarged. This was a shrunken gall-bladder with a stone in it and I removed it. There might have been other stones in the common duct but it was not thickened. There was nothing to feel in it and nothing to justify exploring it: no enlargement, no thickening of its wall and normal bile on aspiration. Even if there is no enlargement and nothing to feel, you still may find, on opening the common duct, a stone or stones. Even when you have opened the common duct you may fail to find a stone. We could find nothing on exploration but even then I was not too confident that I had emptied the duct of any stone. There was no pancreatic lesion. There was a group of enlarged glands at the liver hilum: I took

one out and we learnt nothing from it—there were " simple reactive changes " only. Her recovery from the operation was uninterrupted. She was getting better from her original condition but not because of anything we did to her! She went out of hospital an unsolved problem except that we had excluded any surgical obstruction, so we had not operated unnecessarily. We thought perhaps she was one of those long-standing cases of jaundice due to primary liver disease and that it might possibly clear up.

When this woman came into hospital again (January 2nd, 1954) she was very ill and dying and we could do nothing.

Dr. R. L. Bishton asked Mr. Cooke if he was averse to doing a liver biopsy. Mr. Cooke replied that the liver was very swollen and he was afraid of causing haemorrhage: having done a useless operation, except to exclude anything surgical, he decided not to do a biopsy.

Professor T. F. Hewer: The liver was very swollen?

Mr. R. V. Cooke: It was terribly swollen and looked the liver of an obstructive jaundice; but it must have been a hepatitis.

Professor Hewer asked Dr. J. Guy to tell them something about liver-function tests.

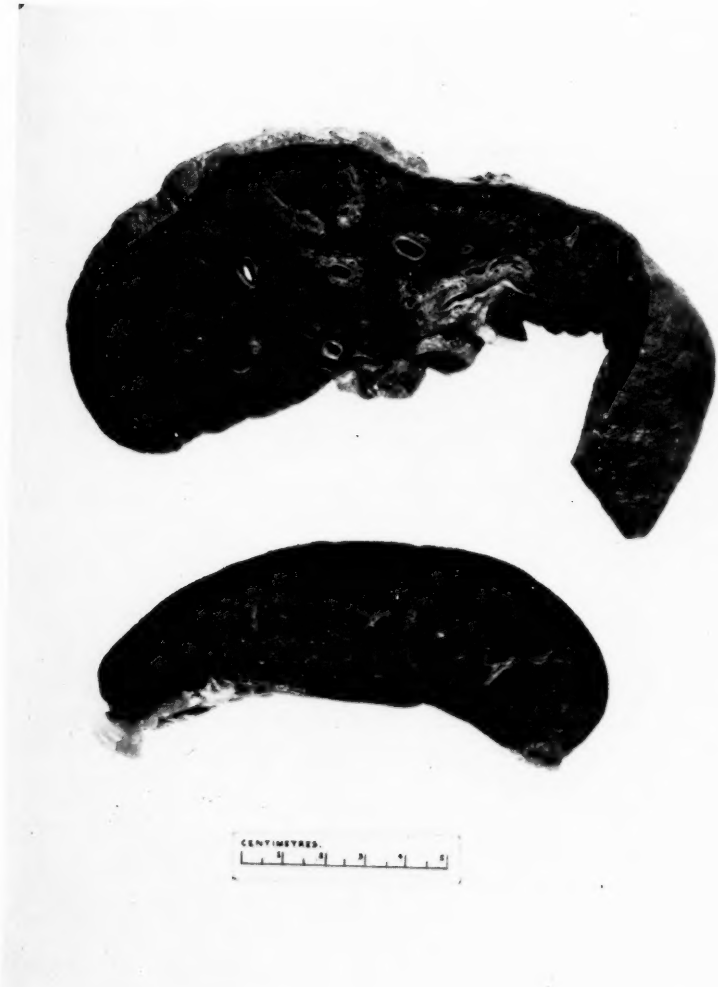
Dr. J. Guy: I think I can agree with Mr. Cooke. I can remember this lady. We had at the time considered whether it was worth doing a test; she had been jaundiced for four to six weeks and the chances of getting an answer were very scanty.

In the early stages of jaundice a distinction can often be made between obstructive and hepatocellular jaundice by the thymol turbidity or flocculation tests, which give higher figures in the latter. In the absence of bone disease, a serum alkaline phosphatase of over 40 units is almost diagnostic of obstructive jaundice, and one of under 15 units of hepatocellular jaundice. In this case the results would probably have been ambiguous.

Dr. O. C. Lloyd (describing the autopsy): This patient was jaundiced. The other external abnormality was the operation wound which had been healing very nicely and was still a purple colour. There was more than a litre of yellow fluid in the abdomen. There were well-marked vascular anastomoses between portal and systemic systems including the adhesions which had been formed by the wound. Blood vessels all stood out in a characteristic pattern and these findings of fluid in the abdomen and dilatation of anastomoses showed that during the course of these three months there had been developing some degree of portal hypertension. The liver was very much smaller than expected and this is interesting in view of the observation at operation that it was enlarged. There was slight roughening of the surface and nodular irregularity. The spleen was much congested and enlarged. The congestion was partly recent and partly chronic. It was a passive venous congestion. (Plate XIX.) The portal hypertension had not been going on long enough to produce fibrosis of spleen. The gall-bladder was missing. The bile-ducts were not dilated or obstructed in any way and there was no evidence of obstruction in the past. The liver showed the changes of early cirrhosis, such as are found after two or three months of an acute hepatitis that has not cleared up. The bile ductules (Plate XX) seemed to be increased in numbers, but this may have been not so much evidence of proliferation, as the result of atrophy of the parenchyma which supported them. Bile thrombi were seen. (Plate XXI). All the cells around the central vein had died. This liver cell death was not the original necrosis when she first had her attack of acute hepatitis. We know from the experience of those who have done serial biopsies on livers, that the necrotic remains of the cells get removed completely in about five or six weeks. This suggested to me that there had been some acute liver cell death within the last four or five days. The reason for this was probably poor blood flow in the central parts of the lobules of the liver.

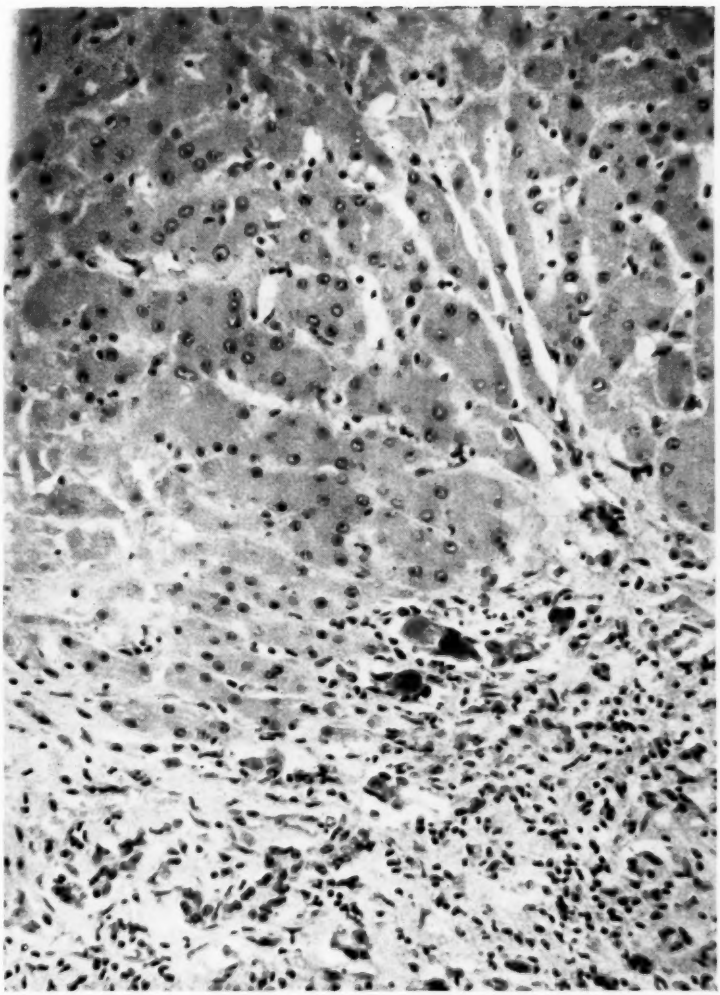
Here is the case of a woman who had acute hepatitis, whose liver was observed to be enlarged after five weeks, whose jaundice did not clear up, who went on to develop a mild degree of portal cirrhosis and who, in consequence of this portal cirrhosis,

PLATE XIX



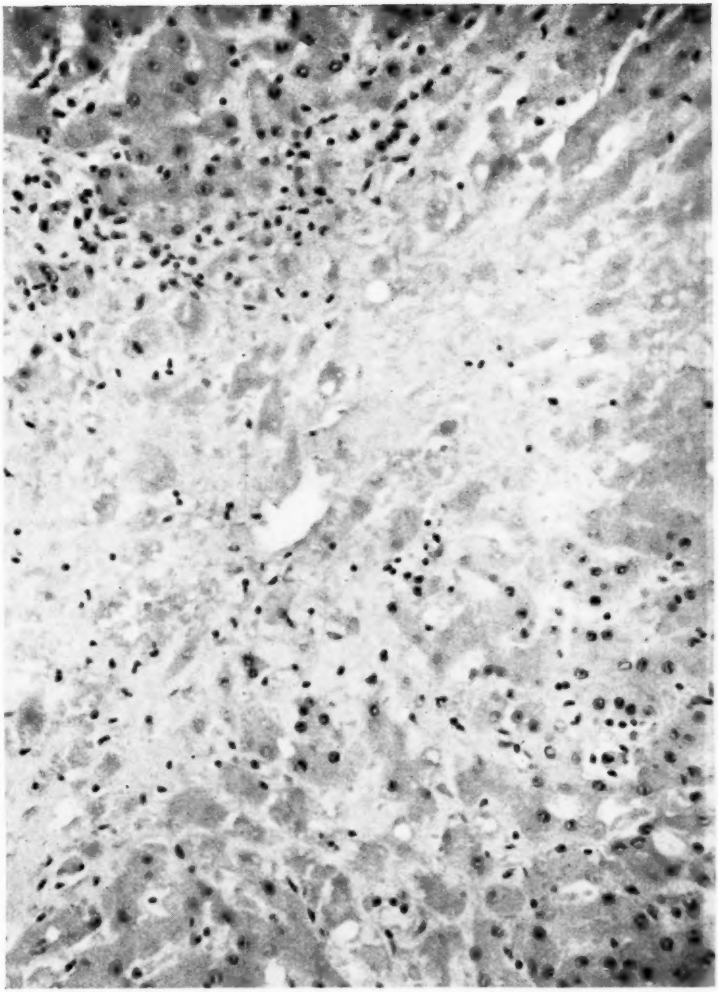
Liver and spleen

PLATE XX



Periphery of liver lobule showing some small bile-ducts dilated with bile (× 220)

PLATE XXI



Centre of liver lobule showing extensive area of necrosis around the central vein (× 220)

developed portal hypertension. I think there is good evidence in this case that the cause of death was hepatic failure.

Mr. R. V. Cooke: Is it just the liver failure that kills them?

Dr. Lloyd: I think it was one of the important causes in her. In acute hepatitis there can be far more necrosis yet they seem to recover because the liver parenchyma has a wonderful power of regeneration.

Professor Hewer: Even in this case, with so much survival of the parenchyma, death was due to hepatic failure. The function of the liver cells must have been seriously impaired by the ischaemia that so recently produced the centrilobular necrosis. Even the regenerated liver cells must have been suffering from severe ischaemia and could not perform their proper function.

A Student: Was a reticulin stain done?

Dr. Lloyd: No, it was not. A reticulin stain gives you a very good idea of the liver lobule architecture. But I did a van Giesen stain which shows the portal fibrosis equally well and also gives distinction between the old colagen fibres and the new ones formed as a result of more recent disease.

Professor Hewer: In addition to the diagnostic interest of this case there is the further, most important, observation that the liver shrank from a state of enlargement, actually observed at operation, to a small cirrhotic organ in only eight weeks. This is in keeping with what we know of some other cases of hepatitis that run an unfavourable course and give rise to portal cirrhosis.

News from Societies

TORQUAY AND DISTRICT MEDICAL SOCIETY

"Collaboration between Church and Medicine"

As a result of a public pronouncement by the Archbishop of Canterbury on the participation of the clergy in the art of healing, it was determined that a meeting of interested members of the respective professions concerned should be held in the South Devon area, not only with a view to discussing the subject itself, but also with a view to taking practical steps both to identify the ground common to the Church and Medicine and to indicate whether co-operation could be effected. Such a meeting was called at the Torbay Hospital on February 26th, 1953, on the initiative of the Torquay and District Medical Society, on which occasion the local clergy joined the Society in the meeting, forty-five clergy and forty-one doctors being present. The Bishop of Exeter opened a discussion on the place of clergy and doctors in Medicine by pointing out that there was a great need for co-operation between clergy and doctors, for they were both serving in the same ministry. Human nature was a unity composed of body and soul, and because of this unity priests and doctors must work together; their spheres might be distinct and each might have his own expert knowledge, but their objectives were closely interrelated. There was a great temptation for both to arrogate to themselves healing power that belonged to God, whereas at best, in both the spiritual and physical sphere, they were merely channels of grace. The first requirement was a profound humility, realizing that their duty was to create conditions favourable for operation of the Divine power. The Bishop then proposed three topics for discussion. In regard to the views he expressed, he said he was speaking for himself and not for the Church. Of euthanasia he said it was wrong to take life; but it was a doctor's duty to relieve unnecessary pain and it might well be that the drug needed to achieve this might shorten life. Of abortion he said that the doctrine of the Roman Church was that from the moment of conception the embryo had equal rights with the mother. He himself thought that the problem hinged on whether we regarded the unborn baby as having a distinct personality. The infant in the womb certainly had a claim to life; but had it a greater claim than its mother? He thought that if there was a good reason for believing that the continuance of pregnancy would be fatal for the mother, to kill the foetus might be the lesser of two evils and therefore justifiable, especially when to do nothing would mean the death of both. On telling the patient that his end was near, the Bishop thought that it was unfortunate that in recent times the clergy had ceased to consider it their duty to include in their ordinary course of religious instruction the preparation for death. It was so much easier to tell the truth to patients so prepared, and so the best use could be made of their last few days of life. It was of the utmost importance that chaplains should be kept informed of those on the danger-list and be allowed free access to those who were likely to appreciate their administrations.

Many clergy and doctors took part in the discussion which continued for about two hours. A strong feeling became evident that some means should be provided for maintaining a liaison between clergy and doctors in the area. The Bishop welcomed this idea and closed the meeting by charging the Rural Dean to collaborate with the Council of the Medical Society in selecting a small committee of clergy and doctors to consider how this purpose could best be achieved.

Such a committee was constituted later and discussions have taken place on subjects of common interest. It was soon evident that the most fruitful field would be that pertaining to the emotional state of the patients' minds and relating to their thought processes. There was a meeting held in April at which the problem of pain was discussed; and at a subsequent meeting the problem of fear was debated. There

were two openers of this discussion: Dr. Guy Tanner (Newton Abbot) and the Rev. Mr. T. P. Vokes-Dudgeon.

Dr. Tanner said: "A famous physician used to say that it was always fear that drove a patient to see his doctor—fear for his future health, fear for his ability to earn his living, perhaps even fear for his life—and that the doctor who failed to recognize and treat this fear was only doing half his job.

"The patient comes to us doctors with a set of symptoms. They may indicate trouble which is neither dangerous nor disabling. On the other hand, they may be the first signs of a downhill journey with little hope of recovery. They may foretell pain, loss of earning power, the prospect of surgical operations. They may be themselves the result of fear as well as its cause. So many people come to us with physical symptoms due solely to mental anxiety, whose real trouble is a failing business or an impending family catastrophe. During the War we met these same symptoms caused by the fear of danger which was only too obvious.

"These, then, are the sort of fears which people have to face. The question is, what can we do to help?

"The best thing we can do, and fortunately it is possible in the great majority of cases, is to remove the fear altogether. We do this by reassuring the patient that his malady is not serious and will soon pass. If we cannot do this, if we know that serious trouble is indeed present, we are faced with a difficult problem and a heavy responsibility and that is to decide how much to tell. Under what circumstances and to what extent, are we justified in concealing from a sick person the danger of his or her condition? I will leave the meeting to discuss this problem amongst others.

"We shall, then, be able to remove fear in many cases. We shall feel justified in some others in reducing fear by hiding or understating the danger. There remain many more in which the danger is only too obvious, and fear is inevitably present. Our task must then be to help the affected person to face his fear, to live with it without being overcome by it, and if possible to conquer it.

"We can help in three ways, and the first is by the use of drugs. We can soothe by various members of the barbiturate family and in extreme cases by morphia, and we can boost morale by benzedrine and its derivatives. The patient himself may use alcohol. I will only say this of drugs in the treatment of fear; they are often useful and sometimes essential in an emergency, to help a patient over a bad patch of trouble; but as a long-term policy they are a confession of failure. Fear is a malady of the mind and spirit, and it is through the mind and the spirit that lasting help must come.

"The second way in which we can help is by tackling the patient's mind. Every general practitioner has to be a practical psychologist of sorts, and we do try to talk our patients into a more calm, patient and optimistic frame of mind in the face of their difficulties and fears. But many states of fear, particularly those which are morbidly exaggerated, do sometimes benefit by treatment by a proper psychiatrist; and in one type of fear, the delusional type, this is quite essential. I mean by this the sort of person who is convinced that his neighbour is dropping bombs through his letter-box or poisoning his drinking-water. His fear is madness and only a psychiatrist can hope to tackle him adequately.

"The third way, and to my mind by far the most important way and the best, is through the patient's spirit. Here the family doctor, if he is willing, can help a great deal, the parish priest or minister much more, and co-operation between doctor and priest most of all. If the patient is known to be a religious man, the approach should be easy. If the doctor is himself a convinced and practising Christian, and especially if he has learnt to talk about his own religious faith naturally and without embarrassment, then again the approach should be easy. If the doctor is shy about religion or the patient antagonistic (and this latter state is extremely rare in my experience) a direct approach may be difficult; but at least the patient's relations should be reminded of the help that a parish priest or minister is ready and anxious to give. The important thing for us doctors to remember is the fact, proved over and over again by history and human experience, that the Christian faith can enable a human being to face the greatest

dangers in peace of mind and relief from fear. Many of us have the great good fortune to know this from personal experience in our own lives or in the lives of those with whom we have had contact. To the sceptics, if there are any present, I would quote a statement by Sir Heneage Ogilvie in a recent article in *The Practitioner*. He says: " The most effective weapon in the treatment of the troubled spirit may be the faith in a power higher than ourselves." This " may be " sounds like the statement of a sceptic who is becoming sceptical of his own scepticism! Anyway, whether we say " may be " with Ogilvie or " is " with conviction, we should at least offer this greatest of all comforts to those who come to us for help. We can do so by recognizing our brothers of the clergy as specialists in faith, and calling in their assistance much more often than we do at present.

The Rev. Mr. T. P. Vokes-Dudgeon, (Vicar of St. Marychurch, Torquay) spoke of fear, and of its evil effects. Religion replaced fear by hope, and even death became less frightening. Mr. Vokes-Dudgeon explained his views on the place of Baptism and the Eucharist in easing man's fear, and how some people might be relieved by confession and absolution. He said that the Sacrament of Unction brought calm to very sick people. No two patients were precisely alike and the priest must use patience and discrimination. Moreover, he should not dabble in the antics of the psychiatrist.

Notes and News

The usual Sunday morning lecture-demonstrations for general practitioners were held from 27th March to 22nd May, 1955.

Intensive week-end courses for general practitioners were held at Exeter from 13th to 15th May, 1955, and at Cheltenham from 24th to 26th June, 1955.

UNIVERSITY OF BRISTOL

Dr. R. F. Woolmer, promoted to a Readership in Anaesthetics.

Dr. T. D. Williams, B.SC., M.B., CH.B., Lecturer Grade II in Physiology, 25th March, 1955.

Dr. J. L. Jarvis, M.B., B.S., L.M.S.S.A., Dr. M. S. Dunnill, M.B., CH.B., Demonstrator in Pathology (2), 10th June, 1955.

Dr. T. M. Abbas, M.B., CH.B., M.D., F.R.C.S., M.R.C.O.G., Lecturer in Obstetrics and Gynaecology, 3rd May, 1955.

DOCTOR OF MEDICINE: Herford, M. E. M., and Schnieden, H.

MASTER OF SURGERY: Slade, P. R.

M.B , Ch.B. (JUNE, 1955)

WITH SECOND CLASS HONOURS

Burge, Elizabeth—with distinction in Public Health.
Moore, Joyce, Esme—with distinction in Public Health.
Polkinghorne, Mavis Mary—with distinction in Medicine.

PASS

Allwood, Geoffrey Keith
Bate, Dorothy Frances
Bebell, Jean Margaret
Beckingham, Prudence Mary
Bolton, Hubert Fairfield
Charleston, Mervyn Threfall
Chesterfield, Margaret Edith
Clarke, John Picton James
Craven, Shirley Ada
Faireman, Edith
Farrelly, Francis Gerald
Foley, David John
Hartwell, James Keith
James, Andrew John Seldon
Jorro, Mary Theresa Catherine Ann
Lambert, Peter Michael
Laurence, Pamela Marjorie
Leighton, Penelope Anne
Mercer, Nancy—*with distinction in Public Health*
Midwinter, Ralph Edward
Naylor, Gerald Alexander
Neilson, Jeffrey Benjamin—*with distinction in Public Health*
Nicholls, Hedley John Leslie
Roylance, Peter James
Simpson, Donald Watson—*with distinction in Obstetrics and Gynaecology*
Smith, Edward Michael
Stewart, Anne Lindsay
Wallace, Edgar

In Group I completing the examination
Keevil, Janet Hazel
McCormick, Robert George

Francis Dudley Memorial Prize for 1954: C. C. Entwistle.
The Crosby Leonard Prize for the Present Session: A. Morgan.
The Dental Gold Medal for 1954: M. D. H. Allen.
The Thomas Francis Edgeworth and Francis Henry Edgeworth Prize in Physiology: P. R. Golding.
Mackie Pathology Prize for 1955: J. H. Williams.
Hewer Pathology Prize for 1954-55: J. A. Hayes.
Tibbits Memorial Prize for 1955: Penelope A. Leighton.

F.R.C.S.: Walder, D. N.
M.R.C.O.G.: Soltau, D. H. K.
D.M.R.D.: Moore, E. W.
D.P.M.: (*Part* 2) *Conjoint:* Galla, T., Joyce, M. H. B., Seager, C. P. and Walker, D. L.
D.P.H.: O'Dea, Margaret M. J., Sefton, Elizabeth M., Speller, P. J. and Reilly, M. D.
D.A.: Forrest, Mari, Christie, Anne W., Haines, Ann M.
New Year Honour—1955: A. F. Alford, M.B., CH.B., C.B.E. (Civil Division).

Appointments

UNITED BRISTOL HOSPITALS

Name	*Appointment*	*Formerly*
CHITTY, K., M.B., B.CH., F.R.C.S.	Senior Registrar in General Surgery, United Bristol Hospitals.	Senior Registrar in Genito-Urinary Surgery, United Bristol Hospitals
AKEHURST, A. C., M.B., B.S., F.R.C.S.	Registrar in General Surgery, United Bristol Hospitals.	Senior House Officer in General Surgery, United Bristol Hospitals.
MCINNES, A. D., M.B., CH.B., M.R.C.P.E.	Senior Registrar in General Medicine, United Bristol Hospitals.	Registrar in Medicine, Western Infirmary, Glasgow.
EDWARDS, E. M., M.B., B.S., M.R.C.O.G.	Senior Registrar in Obstetrics & Gynaecology, Southmead Hospital.	Registrar in Obstetrics & Gynaecology, United Bristol Hospitals.
TREISMAN, O. S., M.B., B.CH., M.R.C.O.G.	Registrar in Obstetrics & Gynaecology, United Bristol Hospitals.	Senior Medical Officer in Obstetrics & Gynaecology, King Edward VII Hospital, Durban.
RICHARD, D. R., B.M., B.CH.	Registrar in Orthopaedics, United Bristol Hospitals.	Registrar in Surgery, Weston-super-Mare General Hospital.
HARDY, H. M., M.B., CH.B., D.A.	Senior Registrar in Anaesthetics, Leeds.	Registrar in Anaesthetics, United Bristol Hospitals.
CHANDLER, C. C. D., M.R.C.S., L.R.C.P., D.A.	Registrar in Anaesthetics, United Bristol Hospitals.	Registrar in Anaesthetics, Kingston Hospital.
SIMPSON, K., M.D., B.S., M.R.C.P.	Registrar in Paediatrics, United Bristol Hospitals.	Registrar to Children's Department, St. Thomas' Hospital.
COLE, MISS P. M., M.B., B.S., D.C.H.	Registrar in Paediatrics, United Bristol Hospitals.	Registrar in Paediatrics, Plymouth.
MCCORMICK, A. J. A., M.B., CH.B., D.O.M.S., F.R.C.S.	Registrar in Ophthalmology, United Bristol Hospitals.	Senior Registrar to Eye Department, Edinburgh Royal Infirmary.
HOLT, J. A. G., M.B., CH.B., F.R.C.S., D.OBST.R.C.O.G.	Registrar in Radiotherapy, United Bristol Hospitals.	Senior House Officer in Radiotherapy, United Bristol Hospitals.
SLADE, N., M.B., CH.B., F.R.C.S.	Senior Registrar in Genito-Urinary Surgery, United Bristol Hospitals.	Surgeon, Port Sudan Civil Hospital.
ST. CLAIR, J. D., M.B., CH.B., D.A., F.F.A.R.C.S.	Registrar in Anaesthetics, United Bristol Hospitals.	National Service, R.A.M.C.
EVANS, W. A., M.B., CH.B.	Registrar in Radio-diagnosis (part-time), United Bristol Hospitals.	Radiologist, Grey Hospital, Greymouth, New Zealand.

SOUTH-WESTERN REGIONAL HOSPITAL BOARD

September 1954 *to June* 1955

BRISTOL

Name	*Appointment*	*Formerly*
WALKER, W. LUMSDEN, M.B., CH.B., D.P.H., D.P.M.	Medical Superintendent, Hortham-Brentry Hospitals Group.	Senior Registrar in Mental Deficiency & Child Psychiatry, Bristol.
MAKSIMCZYK, K., M.B., CH.B. (POLISH SCHOOL OF MEDICINE, EDINBURGH).	Asst. Medical Director, Mass Radiography Units, Bristol (S.H.M.O.).	Asst. Medical Director, Mass Radiography Units, Edinburgh.
WALLEY, R. V., B.A., M.B., B.CHIR.	Deputy Resident Physician (S.H.M.O.), Ham Green Hospital, near Bristol.	J.H.M.O., Ham Green Hospital.
PARTRIDGE, J. P., M.B., B.S., F.R.C.S.	Senior Registrar in General Surgery, Southmead Hospital.	Senior Registrar in General Surgery, United Bristol Hospitals.
WATERFALL, JOAN M., M.B., CH.B., D.A., PRIMARY F.F.A., R.C.S.	Temporary Senior Anaesthetic Registrar, Frenchay Hospital.	Locum Senior Anaesthetic Registrar, The London Hospital.
WEST, MARY F., M.B., B.S.-(LOND), D.A. (PART I).	Anaesthetic Registrar, Southmead Hospital, Bristol.	Locum S.H.O. Anaesthetist, Swansea General Hospital.
FLETCHER, J. P., B.D.S., L.D.S., R.C.S., PRIMARY F.D.S., R.C.S.	Registrar in Dental Surgery, Bristol.	National Service in the Army.
WELLS, A. MARO, M.B., CH.B., M.R.C.S., L.R.C.P., D.OBST., R.C.O.G., D.C.H.	Medical Registrar, Southmead Hospital, Bristol.	S.R.O., Bristol Royal Infirmary, & S.H.O. to Professor Perry & Dr. Cates.
HARGADON, E. J., M.B., B.CH., M.R.C.S., L.R.C.P. PRIMARY F.R.C.S.	Surgical Registrar, Cossham-Frenchay Hospitals Group, Bristol.	Surgical Registrar, Caerphilly & District Miners' Hospital.
LEAHY, T. C., M.B., B.CH., B.A.O.	S.H.M.O., Hortham-Brentry Hospitals Group.	Locum S.H.M.O., Hortham-Brentry Hospitals Group.
BRADFIELD, A. M., M.B., CH.B., (NEW ZEALAND).	Psychiatric Registrar, Bristol Mental Hospitals.	Senior House Physician, St. Benedict's Hospital, Tooting.
GALLA, T., M.B., CH.B., D.P.M.	Senior Psychiatric Registrar, Bristol Mental Hospitals.	Psychiatric Registrar, Bristol Mental Hospitals.
LEIGH, H. I., M.B., CH.B.,	Registrar in the Dept. of Neurological Surgery, Frenchay Hospital, Bristol.	Medical Officer, R.A.F.
EDWARDS, E. M., M.R.C.S., L.R.C.P., M.B., B.S., D.OBST.R.C.O.G., M.R.C.O.G.	Temporary Senior Registrar in Obstetrics & Gynaecology, Southmead Hospital, Bristol.	Registrar in Obstetrics & Gynaecology, United Bristol Hospitals.
JOLLY, G. F., M.R.C.S., L.R.C.P., M.A., M.B., B.CH., D.OBST.R.C.O.G., M.R.C.O.G.	Registrar in Obstetrics and Gynaecology, Southmead Hospital, Bristol.	Locum Registrar to the Professorial Unit, United Birmingham Hospitals.
STEWART, R. L. N., M.B., CH.B., D.O.	Opthalmologist, Bristol, (S.H.M.O.).	Senior Resident at Bristol Eye Hospital.
BROCK, B. H., M.R.C.S., L.R.C.P., M.B., B.CHIR., F.R.C.S.	Orthopaedic Registrar, United Bristol Hospitals and Winford Orthopaedic Hospital.	Orthopaedic Registrar, St. Olave's Hospital, Rotherhythe.
FULLER, ANGELA D., M.R.C.S., L.R.C.P., M.B., B.CHIR., D.C.H.	Paediatric Registrar, Southmead Hospital, Bristol.	R.M.O,, Infectious Diseases Unit, Southampton Chest Hospital.
GORDON, A., M.B., CH.B.	Registrar to the Department of Pathology, Frenchay Hospital, Bristol.	S.H.O. to the Department of Pathology, Frenchay Hospital.
POLLARD, B. R., M.R.C.S., L.R.C.P., M.B., B.CHIR.	Assistant Pathologist, Frenchay Hospital, Bristol.	Senior Registrar to Department of Clinical Bacteriology, Wright-Fleming Institute, St. Mary's Hospital.
DOYLE, F. H., B.SC., M.B., CH.B.	Part-time Registrar in Radio-diagnosis, Bristol Clinical Area.	S.H.O. in General Surgery, Maryfield Hospital, Dundee.
SNELL, F. W. G., M.B., CH.B.	Part-time Registrar in Radio-diagnosis, Bristol Clinical Area.	Surgical Registrar, Jersey General Hospital.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
FEARNLEY, G. R., M.R.C.S., L.R.C.P., M.B., B.S., M.R.C.P., M.D.	Consultant Physician, North Gloucestershire Clinical Area.	Lecturer in Medicine, Postgraduate Medical School of London, and Hon. Consultant Physician, Hammersmith Hospital.
THEOBALDS, J. R., M.R.C.S. L.R.C.P.	Medical Registrar, Cheltenham General Eye & Children's Hospital.	Medical Registrar, Royal Salop Infirmary, Shrewsbury.
VILLET, D. DE G., M.B., CH.B., (CAPE TOWN), F.R.C.S.	Surgical Registrar, Gloucestershire Royal Hospital, Gloucester.	Locum Surgical Registrar, Lambeth Hospital.
HAROON, S., M.B., B.S. (PUNJAB), L.M., D.G.O.,	Registrar in Obstetrics and Gynaecology, Gloucestershire Royal Hospital, Gloucester.	Gynaecological House Surgeon, City General Hospital, Gloucester.

BATH

Name	*Appointment*	*Formerly*
SCHOFIELD, T. L., M.B., M.R.C.S., L.R.C.P., F.R.C.S., CH.M.	Consultant Surgeon, Bath, Clinical Area.	Lecturer in Surgery & 1st Asst. to the Professor of Surgery, Liverpool University. Hon. Consultant Surgeon, Broadgreen Hospital, Liverpool.
JONES, E. R., M.R.C.S., L.R.C.P., D.P.M.	Assistant Psychiatrist, Mendip Hospital, Wells.	Assistant Psychiatrist, Cefn Coed Hospital, Swansea.
KNUTZEN, J. S., M.B., CH.B., (CAPE TOWN).	E.N.T. Registrar, Bath Group of Hospitals.	House Surgeon, Royal United Hospital, Bath.
SAXTON, H. M., M.B., B.S.	Medical Registrar, St. Martins Hospital and Royal National Hospitals for Rheumatic Diseases, Bath.	Medical Superintendent, St. Mary's Hospital, London.
HIBBARD, B. M., M.B., B.S., M.R.C.S., L.R.C.P., D.OBST.-R.C.O.G.	Registrar in Obstetrics and Gynaecology, Bath Group of Hospitals.	House Physician, Royal Northern Hospital, London.
NEATBY, G. M., M.R.C.S., L.R.C.P., D.O.M.S., B.A., M.A.	Clinical Asst. in Ophthalmology, Bath Eye Infirmary—one session in the S.H.M.O. grade.	Is in Ophthalmic practice, Bristol.
TUPMAN, G. S., B.SC., M.B., CH.B., F.R.C.S.	Senior Orthopaedic Registrar, Bath Clinical Area.	Orthopaedic Registrar, Lord Mayor Treloar Orthopaedic Hospital, Alton.

SOUTH SOMERSET

Name	*Appointment*	*Formerly*
NELSON, M. P., B.A., M.R.C.S., L.R.C.P., D.P.M.	Medical Superintendent, Sandhill Park Hospital Group, Bishops Lydeard.	S.H.M.O., Harperbury Hospital near St. Albans.
HUBBARD, ST. C. M. L'A., M.R.C.S., L.R.C.P., M.B., B.S.	Clinical Assistant in Orthopaedic & Traumatic Surgery, Taunton & Somerset Hospital. (1 session).	Is in general practice in Taunton.

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
DAWSON, J. J. Y., M.R.C.S., L.R.C.P., M.B., B.S., M.D., M.R.C.P.	Senior Consultant Chest Physician, Plymouth Clinical Area.	Medical Chief Assistant, Brompton Hospital.
EARLE, B. V., M.B., B.CH., B.A.O., M.R.C.P., M.D., D.P.M.	Consultant Psychiatrist, Exeter Clinical Area.	Locum Consultant Psychiatrist, Powick Hospital, Worcester.
MOORE, H. D., M.B., B.S., (SYDNEY), F.R.C.S.,	Consultant Surgeon, Exeter Clinical Area.	Senior Surgical Tutor, University of Leeds.
MOYLE, I. R., M.A., M.R.C.S., L.R.C.P., D.M.R.D.	Consultant in Radio-diagnosis, Plymouth Clinical Area.	Registrar, Dept. of Radio-diagnosis, United Bristol Hospitals.
MACPHERSON, J., M.B., CH.B., F.R.C.S., M.R.C.O.G.	Consultant Obstetrician and Gynaecologist, Torquay.	Senior Registrar in Obstetrics & Gynaecology, Mill Road, Maternity Hospital, Liverpool.

DEVON AND CORNWALL—continued.

Name	*Appointment*	*Formerly*
PAYNE, I. W., M.B., CH.B., D.O.M.S., F.R.C.S.	Consultant Ophthalmologist, Plymouth Clinical Area.	Senior Registrar, Royal Eye Hospital, London, S.E.1.
STORMONT, H. A. W., M.R.C.S., L.R.C.P.	General Practitioner Anaesthetist, Ilfracombe & Dist. Hospital. (1 session).	Is in general practice in Ilfracombe.
WINFIELD, A. G., M.R.C.S., L.R.C.P., M.B., B.S.	General Practitioner Anaesthetist, Tavistock Hospital. (1 session).	Is in general practice in Tavistock.
ISAAC, D. H., M.R.C.S., L.R.C.P., M.B., B.S., M.D., M.R.C.P.	Senior Medical Registrar, South Devon & E. Cornwall Hospital, Plymouth (Joint appt. with the United Bristol Hospitals).	Medical 1st Assistant, Sheffield Royal Infirmary.
PYBUS, P. K., M.B., B.CHIR., M.R.C.S., L.R.C.P., D.OBST.-R.C.O.G., PRIMARY F.R.C.S.	Surgical Registrar, Torquay and Newton Abbot Hospitals.	Locum Surgical Registrar, Poplar Hospital, London.
STRANGE, D. M., M.A., L.S.A.	S.H.M.O., Royal Western Counties Institution, Starcross.	J.H.M.O., Mendip Hospital, Wells.
ROBERTS, A. G., B.SC., M.B., B.CH.	Psychiatric Registrar, Moorhaven Hospital, Ivybridge.	S.H.O. in Psychiatry, Whitchurch Hospital, Cardiff.
REILLY, M. C. T., M.B., B.S., M.R.C.S., L.R.C.P., F.R.C.S., M.S.	Consultant Surgeon, Plymouth Clinical Area.	Surgical 1st Assistant, The London Hospital.
GALLAGHER, M. J., M.B., B.S. (QUEENSLAND), F.R.C.S.	Orthopaedic Registrar, Princess Elizabeth Orthopaedic Hospital, Exeter.	S.H.O. Princess Elizabeth Orthopaedic Hospital.
COLE, PAULINE, M.B., B.S., D.C.H.	Registrars in Paediatrics to the South Devon and East Cornwall Hospital, Plymouth, and the United Bristol Hospitals (interchange appointments).	Registrar in Paediatrics, Queen Elizabeth Hospital for Children, Hackney.
LLOYD, JUNE K., M.B., CH.B, M.R.C.P.		S.H.O. in General Medicine, United Bristol Hospitals.
CHUDECKI, B., M.B., CH.B., (CRACOW), D.M.R.T.	Assistant Radiotherapist, Exeter Clinical Area.	Registrar to Radiotherapy Dept. Charing Cross Hospital.
MAINPRISE, J. V., M.B., CH.B.	Assistant Venereologist, Torquay. (2 sessions).	Undertaking same post on locum basis.

ELSEWHERE

Name	*Appointment*	*Formerly*
HAWKINS, J. R., M.B., D.P.M.	Consultant Psychiatrist, Winterton Hospital, Sedgefield, Co. Durham.	—
FRANCIS, M. J., M.B., D.C.H., D.A.	Junior Resident Anaesthetist, Hospital for Sick Children, Gt. Ormond St., London, W.C.1.	—
BOSE, S. N., D.A.	Senior Lecturer in Anaesthetics, Kitchener School of Medicine, Khartoum.	—
PERSEY, DOROTHY J., M.B. BRISTOL, D.C.H.	Asst. Maternity and Child Welfare M.O. and School M.O., Derbyshire.	—
LAURANCE, B. M., M.B., B.S., M.R.C.P., D.C.H.	Consultant Paediatrician, Sheffield Regional Hospital Board.	Senior Registrar in Paediatrics, United Bristol Hospitals.
LEATHER, H. M., M.B., B.CH., M.R.C.P.	First Assistant in Medicine, Makerere College, Uganda.	Senior Registrar in General Medicine, United Bristol Hospitals.
FREEMAN, R. H., M.B., CH.B., F.R.C.S.	Senior Registrar in Orthopaedics, King's College Hospital.	Registrar in Orthopaedics, United Bristol Hospitals.
MARCH, H. F., M.B., B.S., M.R.C.P., D.M.R.D.	Consultant Radiologist, Leeds Regional Hospital Board.	Senior Registrar in Radiodiagnosis, United Bristol Hospitals.